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Table of Contents on 3rd Cover

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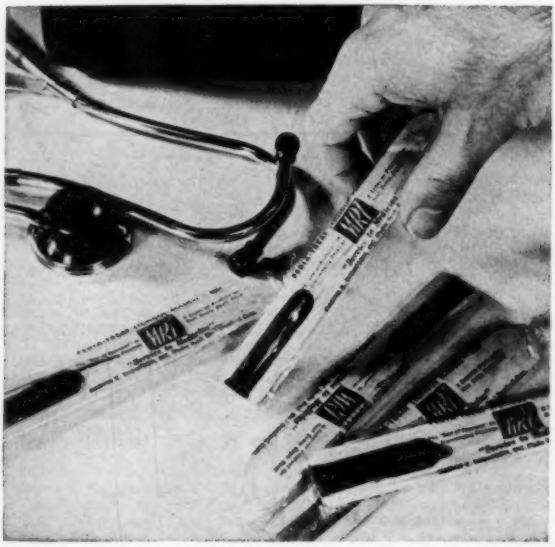

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The Journal of Pediatrics

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BORIC ACID POISONING

REPORT OF FOUR CASES AND A REVIEW OF 109 CASES FROM THE WORLD LITERATURE

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MONTREAL, QUEBEC

BORON compounds have been widely used in medical practice for over a century. Originally used as oral medications, and later as irrigants for every accessible body cavity, they now survive in therapeutics in many forms of topical applications, usually with the illusion of antisepsis. The abandonment of boric acid as an irrigant and as an oral medication undoubtedly resulted from the considerable number of fatalities due to such usage recorded early in this century.

In spite of a steadily growing accumulation of literature concerning the dangers of boric acid, the drug continues to enjoy a widespread and totally undeserved popularity, particularly in pediatric practice.

The historical evolution of topical agents in therapy often reveals that the chief secret of survival in usage is not so much the power to do good as the failure to do harm. In both respects, boric acid has been tried and found wanting.

From the Departments of Pediatrics, The Children's Memorial Hospital and McGill University.

Presented at the meeting of the Canadian Pediatric Society, June 11, 1953, at Quebec City, Canada.

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HISTORICAL

Boric acid was first introduced by Wilhelm Homberg (1672-1715)[40] who prepared the substance by the action of mineral acids on borax. The earliest boric acid medication was known as sal sedativum Hombergi.

Fatal cases of poisoning are reported in the literature as early as 1881. Shoemaker,[41] in 1905, quoted a M. Chevalier who had collected twenty-two cases from the literature. The first good clinical description of poisoning in an infant was recorded by McWalter[10] in 1907. He concluded that the evidence of poisoning in his case was "as conclusive of the evil effects of the drug as a single instance can possibly be. . . ." A 2-month-old infant had been treated for thrush by applications of borax and honey (mel boracis) to the mouth. The infant seemed to enjoy the medication, and the mother continued to apply it from the second to the eighth week of life. McWalter's clinical description is worthy of quotation:

"During this time a progressive wasting had set in, and when I saw the

infant there was a marked erythematous eruption on the palmar aspects of the hands and on the plantar aspect of the feet, with distinct desquamation between the toes and fingers; well-marked urticarial eruption was present on the arms and forearms, but the region between the legs was notably free of eruption. There were tumefaction of the abdomen, a raw, pinky redness of the lips, tongue, palate and throat, with vomiting and looseness of the bowels. . . . On stopping the borax and confining the infant to the breast milk, together with a little raw beef juice, it appears to be recovering rapidly."

There have been several reports of boric acid poisoning by accidental ingestion. Some of these have occurred in newborn nurseries, where boric acid solution was mistakenly used in the preparation of feedings. Other cases have followed its accidental use in hypodermoclyses and intravenous infusions. Such episodes have resulted in the prohibition of use of the drug for any purpose in many maternity and newborn services.

Aikman[22] has referred to a fatal case in a 2-day-old nursing infant, whose mother's nipples had been cleansed with boric acid solution.

There have been several excellent reviews on the subject of boric acid poisoning, notably those of Ross and Conway,[29] McNally and Rukstinat,[34] Fellows and associates,[35] Pfeiffer,[39] and of Brooke and Boggs.[46] Pfeiffer and associates[42] have also contributed valuable experimental data on the toxicology of boric acid.

We have been able to find 105 cases of boric acid poisoning reported in the literature to date and wish to add four cases which have come under our observation. Although some of the reported cases are lacking in detailed description, there is ample evidence upon which a distinctly recognizable clinical picture of acute poisoning may be established and correlated with experimental and pathological findings.

The following four cases are presented to illustrate the hazards associated with the use of topical boric acid preparations in young infants:

CASE REPORTS

CASE 1.—J. G., an 18-day-old female infant, was admitted to the Children's Memorial Hospital on July 15, 1952, with a history of intermittent diarrhea and vomiting since 2 days of age. Birth weight was 6 pounds 5 ounces. Weight on admission was 5 pounds 7 ounces. The mother stated that the infant seemed to have pain on micturition.

Physical Examination.—Physical examination revealed moderate dehydration with very severe excoriation of the buttocks and perineum. There was marked erythema of the face, hands, and feet. The pharynx was slightly injected. Remainder of physical examination was essentially negative.

Laboratory Findings.—Hemoglobin was 16.5 grams, white blood count 6,200, with slight neutropenia. Urinalysis and blood Wassermann test were negative. Carbon dioxide combining power, chloride, and total proteins were within normal limits. Throat and stool cultures showed no pathogens.

Course in Hospital.—Because of the severe excoriation of the buttocks associated with erythema of the face and extremities and with intractable diarrhea and vomiting, the possibility of boric acid intoxication was considered soon after admission. The mother subsequently revealed that, for several days prior to admission, she had applied starch and boric acid powder directly to the buttocks several times daily.

A catheter specimen of urine showed positive qualitative and confirmatory turmeric tests for boric acid.

The infant was treated with intravenous infusions and penicillin intramuscularly. Severe diarrhea persisted for the first three days in hospital and gradually diminished over the next five days. There was intermittent vomiting of small amounts during the first week, after which the infant began to take feedings fairly well, and to gain weight. The early course was complicated by the development of an abscess in the right buttock at the site of a penicillin injection and of coincident otitis media. These infections cleared rapidly on terramycin therapy.

The excoriation of the buttocks improved very slowly with exposure to air. The erythema of the face and extremities cleared over the first week in hospital, showing desquamation. The child was discharged, much improved, sixteen days after admission, weighing 5 pounds 11 ounces.

CASE 2.—M. M., a 20-day-old male infant, was admitted to the Children's Memorial Hospital on Jan. 30, 1953. The child had been born at home, of a normal pregnancy and delivery. Birth weight was 5 pounds. The mother stated that at 3 days of age the child developed loose green stools. His food intake had been rather poor. Five days prior to admission, the infant developed excoriation of the buttocks. From this time until the day of admission, the mother had applied boric acid powder directly to the buttocks on numerous occasions. For several days prior to admission, the infant had been having episodes of stiffening of the entire body several times daily. On the morning of admission, the child developed a generalized erythematous rash and had several episodes of cyanosis. The mother noted that the infant had had projectile vomiting after every feeding for the preceding two days.

Physical Examination.—On admission, physical examination revealed a moderately dehydrated premature infant weighing 4 pounds 13 ounces. The entire body was covered with an irregular erythematous maculopapular rash, most marked on the face, chest, abdomen, and arms. The buttocks and perineum were raw and intensely excoriated. The periumbilical area was reddened, and there were multiple areas of excoriation on the neck and arms.

The anterior fontanel was slightly full. There was bilateral conjunctivitis without discharge. The eyelids were swollen. There was mild bilateral otitis media. The lips, oral mucous membranes, tongue, and palate and pharynx were inflamed, with pinpoint areas of ulceration. Tendon reflexes were hypoactive. There was a weak Moro reflex. The infant had periods of unconsciousness lasting 2 to 3 minutes four to six times per hour. Temperature was normal. Pulse was 150 per minute and regular.

Laboratory Findings.—Hemoglobin was 17.2 grams, white blood count 30,000 with neutrophilia. Urinalysis revealed 2 to 4 white blood cells per high power field. Cultures of the nose, throat, blood, stool, and cerebro-spinal fluid showed no pathogens. Lumbar puncture revealed clear fluid, normal pressure. There were 8 white blood cells per cubic millimeter, 7 lymphocytes, 1 polymorphonuclear. Cerebrospinal fluid protein was 47.4 mg. per cent.

Course in Hospital.—Because of the strongly suspicious history and physical findings, both urine and cerebrospinal fluid were immediately tested for the presence of boric acid. The urine gave a very strongly positive test with turmeric paper, and the spinal fluid gave a definite, though less strongly positive, test. The child was given intravenous infusions, and started on procaine penicillin 300,000 units daily plus streptomycin 75 mg. b.i.d. intramuscularly. The skin was treated by exposure to air. Diarrhea and vomiting gradually subsided over the first five days in hospital. Two days after admission there began a desquamation which rapidly extended over the entire body. The infant literally shed his skin in large sheets.

The erythema gradually disappeared during the first week in hospital, but desquamation continued over a period of eleven days. There were no convulsive manifestations or periods of unconsciousness following the day of admission.

Daily urine tests with turmeric paper showed boric acid to be present in decreasing amounts for the first four days in hospital; following this time, only faint traces and negative tests were obtained. Simultaneous tests were done daily on control urine from other infants on the ward (all negative) and with known boric acid solutions, to assure the sensitivity of the paper. The infant's course was one of intermittent improvement, his progress being impeded by the development of paronychia on one of the desquamating fingers, and later by slow feeding and a tendency to regurgitate unless given small amounts frequently. Fluoroscopic and x-ray examination of the upper gastrointestinal tract on February 26 failed to reveal any abnormality. An electroencephalogram on March 5 was considered normal for the child's age.

He finally began to take and retain increasing amounts of food, and to show a slow but progressive weight gain. He was discharged, much improved, on March 13, 1953, weighing 5 pounds 13 ounces.

CASE 3.—J. S., a 13-day-old male infant, was admitted to the Children's Memorial Hospital on Feb. 5, 1953, with the following history: The child had been born approximately 6 weeks prematurely, a difficult breech delivery, with placenta previa. Birth weight was 5 pounds 8 ounces. The infant required resuscitation at the time of delivery, but afterward did fairly well and was discharged on the eighth day, weighing 5 pounds.

Beginning on the ninth day of life, the child developed diarrhea, with fifteen to twenty liquid stools per day. Two days before admission, he developed excoriation of the buttocks which were treated with repeated applications of boric acid powder. The child began to vomit his feedings. The mother noted that the excoriation became worse, and that an intensely red eruption appeared on the chest and abdomen. On the night before admission, frequent twitchings of the face and extremities were noted. The infant was seen at home by a physician who suspected boric acid poisoning and arranged admission to hospital.

Physical Examination.—Physical examination revealed an extremely undernourished and dehydrated infant. There was a confluent, intensely red eruption over the ears, chest, abdomen, and axillary regions, with desquamation on the upper chest and in the periumbilical region. The buttocks were severely excoriated, red, and edematous. The conjunctivae were injected. His cry was strong. Weight on admission was 4 pounds 5 ounces. Temperature was 97° F.

The anterior fontanel was full and moderately tense; questionable bilateral Kernig. Remainder of the examination was essentially negative.

Laboratory Findings.—Hemoglobin was 13.7 grams. White blood count was 18,000 with slight neutrophilia. The cerebrospinal fluid was turbid and loaded with red blood cells. There were 11 white blood cells; 3 polymorphonuclears, 8 lymphocytes, Pandy positive. Cerebrospinal fluid protein: 85.5 mg. per cent, sugar 120 mg. per cent and chlorides 428 mg. per cent.

Stool culture revealed the usual fecal flora; light growth of *Proteus mirabilis;* light growth of *Staph. pyogenes.* Urine (catheter specimen) showed very strongly positive turmeric paper test for boric acid. Cerebrospinal fluid showed a weakly positive test for boric acid.

Course in Hospital.—Because of the difficult delivery and full fontanel, the possibility of associated birth injury was suspected. Lumbar puncture showed the presence of blood in the spinal fluid.

The infant showed some initial improvement with an intravenous in-

fusion of plasma. However, a few hours after admission the child became cyanotic, with shallow irregular respiration. His condition rapidly deteriorated, and he died approximately ten hours after admission. Autopsy was performed under the supervision of Dr. F. W. Wiglesworth.

Post-mortem Findings.—The lungs grossly were markedly overweight and showed an extensive hemorrhagic consolidation. However, microscopically, the consolidation was seen to be due to widespread recent intraalveolar hemorrhage. Red blood cells, fibrin, and mucus were present in many bronchi and bronchioles. Nowhere was there evidence of inflammation. The cause of the extensive intrapulmonary hemorrhage could not be determined.

The brain showed an extensive subarachnoid hemorrhage (mainly on the left side) with a tear of the right leaf of the tentorium cerebelli. A large subdural clot was present subtentorially on the right. This hemorrhage probably occurred at the time of birth as there was evidence of organization and numerous hemosiderin-laden macrophages.

The other major gross lesions were those of the skin, as described clinically, and a left otitis media. Detailed histologic examination of all organs showed no significant or specific lesions which might be related to boric acid poisoning.

CASE 4.—R. R., a male infant, aged 15 days, was admitted to Children's Memorial Hospital, March 22, 1953. He was born at home at full term. Complaints were of persistent diarrhea and vomiting beginning shortly after birth. Beginning on the second day of life, the mother had applied a well-known brand of baby powder (known to contain boric acid) very liberally to the body.

On admission, the infant was malnourished and dehydrated. Buttocks were very excoriated, red, and boggy. There was intense erythema, most marked on the lower trunk and legs. The cry was high-pitched, and there was slight head retraction.

Laboratory Findings.—Hemoglobin was 14.4 grams. White blood count was 36,000, with neutrophilia. Urine revealed albumin 200 mg.; an occasional white blood cell. There were many granular casts. Cerebrospinal fluid revealed clear fluid; 1 red blood cell; 3 white blood cells; Pandy positive. Cerebrospinal fluid protein showed 107.6 mg. per cent. Urine and cerebrospinal fluid both showed positive turmeric paper tests for boric acid.

Course in Hospital.—Diarrhea and vomiting continued over the first nine days in hospital and then gradually subsided, with gradual weight gain. Twenty-four hours after admission, there began a widespread desquamation of the erythematous areas, involving the buttocks and the entire surface of the legs.

Boric acid was detected in decreasing amounts over the first four days in hospital, and subsequent tests were negative. Cerebrospinal fluid gave a negative test on the fifth day. The infant developed some secondary staphylococcal skin infections which cleared with antibiotic therapy. He subsequently gained weight, remained asymptomatic, and was discharged.

REVIEW OF CASES FROM THE WORLD LITERATURE

Including the four cases reported above, we have been able to find 109 cases of boric acid poisoning reported in the literature.[1-39] Post-mortem findings are recorded in twenty-seven cases, and, in several instances, quantitative determinations of the boric acid content of various organs and tissues have been reported.

Age.—Ages ranged from newborn infant to 72 years of age. Thirty-eight patients, or 34.9 per cent, were under 1 year of age. In this age group the

mortality was 70.2 per cent, as compared with 55.0 per cent for the entire series.

Route of Intoxication.—The various routes of intoxication, with mortality in each group are summarized in Table I.

Signs and Symptoms.—Adequate descriptions of signs and symptoms were recorded in eighty cases. The commonest clinical findings were referable to the nervous system, gastrointestinal tract, and skin.

Symptoms referable to the gastrointestinal tract were found in 73 per cent of this group. Vomiting and diarrhea were more common, and abdominal pain was recorded as a symptom in three adults. In a few instances, the vomiting was described as projectile.

Nervous system symptoms were present in fifty-three patients, or 67 per cent of the group. In younger patients the common findings were those of meningeal irritation, with convulsions, delirium, and coma appearing frequently. Often there was progression to cyanosis and collapse. In adults, headache, marked weakness, and excitement or depression have been reported.

Skin manifestations were striking and were fairly constant in pattern. Eruptions were present in 76 per cent of this series and in 88 per cent of children under 2 years. The typical findings consisted of an intense erythematous eruption, often covering the entire body, followed in one or two days by extensive desquamation. The rash has been variously described as erythematous and confluent, maculopapular, urticarial, or scarlatiniform. The palms and soles were often particularly red. Birch[21] describes the fingers in his patient as being "red, as if painted." Watson[32] speaks of the "boiled lobster" appearance of the body.

TABLE I. ROUTES OF INTOXICATION WITH BORIC ACID, WITH GROUP MORTALITY

ROUTE OF INTOXICATION	NUMBER OF CASES	NUMBER OF DEATHS
Oral ingestion	51	27
Gastric lavage	4	3
Rectal washes and enemas	7	1
Bladder irrigations	4	2
Subcutaneous clyses	5	2
Vaginal packs	3	0
Intravenous infusions	2	1
Irrigation of empyema cavities	5	5
Surface applications (burns, wounds, skin eruptions)	28	19
Total:	109	60 (55%)

It is of interest to note, in the cases reported by Young and co-workers,[37] that the erythema was particularly prominent on the buttocks and scrotum, although the route of poisoning was *not* by local application but by accidental ingestion!

Petechiae are reported in three infants, all of whom died. Jaundice was noted in two newborn infants, both of whom died. In one of these, postmortem examination was done, but no abnormality was demonstrated in the liver. Therefore, although boric acid is known to be concentrated in the liver, a specific ieterogenic action on a

toxic basis could not be presumed in these infants.

Mucous membranes often shared in the general erythema, especially in young infants, in whom the mouth, pharynx, and conjunctivae were most often inflamed.

The only cardiovascular signs reported are those of weak, rapid pulse and cyanosis. In most cases, these findings were associated with collapse and were probably on a neurotoxic basis.

A few cases showed oliguria and one anuria, and in one of our cases, there was apparent pain on urination.

Table II illustrates the increased incidence of major signs and symptoms in children under 2 years in this series.

TABLE II. SIGNS AND SYMPTOMS IN EIGHTY CASES OF BORIC ACID POISONING

	SKIN RASHES (%)	GASTRO-INTESTINAL SYMPTOMS (%)	C.N.S. SIGNS AND SYMPTOMS (%)
Over 2 yr. (39 cases)	78	65	62
Under 2 yr. (41 cases)	88	95	83

Laboratory Findings.—Very few of the individuals in this series had extensive laboratory investigations. In five patients, lumbar puncture was done and showed no increase in cells. Two cases showed an elevation of cerebrospinal fluid protein. No specific morphological or biochemical abnormalities of the peripheral blood are reported. In occasional cases, the presence of albumin, red blood cells, and leukocytes in the urine was noted.

Post-mortem Findings.—Of the sixty fatal cases in this series, twenty-seven included autopsy findings of sufficient detail to permit evaluation. There appear to be no gross or microscopic changes in any of the tissues or organs which might be considered of specific diagnostic value. The most striking changes were found in the gastrointestinal tract, kidneys, liver, brain, meninges, and skin. Three instances of hydropericardium, one of hydrothorax, and one of hydrops ascites are recorded. Three cases showed hemorrhagic cystitis. A single autopsy in this series was reported to show no significant change in any of the organs.

Changes in the central nervous system consisted of edema and congestion of the brain and meninges; these were the most common findings. Three cases showed diffusely scattered perivascular hemorrhages in the medulla, mid-brain, cerebellum, corpus striatum, tuber cinereum, and lateral hypothalamus.

Alterations described in the liver included enlargement, vascular congestion, fatty change of varying degrees, swelling and granular degeneration of the liver cells, and, in two patients, acute parenchymatous degeneration. Pathologic alterations in hepatic structure were found in thirteen of the twenty-seven autopsies.

The most common finding in the gastrointestinal tract was of an inflammatory nature, with congestion, edema, and exfoliation of the mucosal surfaces. These changes have been described in the esophagus, stomach, small and large bowel. In four cases, enlargement of the mesenteric nodes was reported. Seventeen patients in this series showed some degree of these alterations in the gastrointestinal tract.

Eighteen cases showed significant pathological findings in the urinary tract. Of these, three showed hemorrhagic cystitis. The remainder had

renal changes varying from gross pallor of the kidneys to cloudy swelling and granular degeneration of tubular cells. One case showed diffuse cortical degeneration involving the glomeruli and proximal tubules, while, in another, there were bilateral cortical hemorrhages.

From these findings, it may be seen that, in general, the pathologic changes produced by intoxication with boric acid are consistent with the effects of a chemical irritant. These effects are most prominent at the site of application of the poison, at the sites of excretion (urinary tract, gastrointestinal tract, and skin) and of maximum body concentration (brain and liver).

TABLE III. CORRELATION OF EXPERIMENTAL, PATHOLOGICAL, CLINICAL AND LABORATORY FINDINGS IN BORIC ACID POISONING

ORGAN OR SYSTEM INVOLVED	EXPERIMENTAL FINDINGS	HISTOPATHOLOGICAL CHANGES IN MAN	SIGNS AND SYMPTOMS	LABORATORY FINDINGS
Central nervous system	1. Highest organ concentration in the body 2. Neuronophagia, round cell infiltration, and hyperchromatosis 3. Displacement of phosphorus in brain by boron	1. Congestion and edema of brain and meninges 2. Scattered perivascular hemorrhages	Excitement or depression Headache Weakness Signs of meningeal irritation Coma or delirium Convulsions Collapse and cyanosis	Boric acid in cerebrospinal fluid (turmeric test)
Gastrointestinal tract	Small amount excreted by gastrointestinal tract	Vascular congestion Enlarged mesenteric nodes Exfoliative gastroenterocolitis	Vomiting Diarrhea Occasional crampy abdominal pain	Small amounts of boric acid may be demonstrated in feces
Urinary tract	80 to 100% excreted in urine Glomerular and tubular drainage with cell degeneration and debris	Cloudy swelling and granular degeneration of tubular cells Rare cortical degeneration Occasional hemorrhagic cystitis	Diminished urine output Rare anuria ? Pain on micturition	Boric acid in urine Occasional red blood cells, white blood cells, and albumin in urine
Liver	Second highest organ concentration in body Minimal histological changes	Congestion Fatty change Rare parenchymatous degeneration	? Rare jaundice	None recorded
Skin	Small amounts excreted in sweat and saliva	Exfoliative dermatitis with loss of keratin layer	Intense erythema with macules and/or papules. Followed by desquamation Rare petechiae	

In the individuals demonstrating skin eruptions, the findings were those of an exfoliative dermatitis, with desquamation of the stratum corneum, and round cell infiltration of the malphigian layer.

Chemical Analyses and Experimental Toxicity.—Reports of chemical analyses of body organs for boric acid were found in fifteen cases. The re-

sults of these studies are essentially in agreement with those of experimental poisoning in animals. The highest concentrations of boric acid are usually found in the brain and liver, with lesser amounts in the bowel, heart, lungs, stomach, and kidneys.

McNally and Rust[20] recorded average concentrations of 0.210 per cent in the brain, and 0.182 per cent in the liver, in six fatal cases. Pfeiffer and associates[42] have shown a similar distribution of the poison in dogs, and have also demonstrated a high concentration in body fat. This latter observation is not surprising in view of the increased solubility of boric acid in glycerol.

The extremely high degree of absorption of boric acid from broken skin surfaces, serous cavities, and the gastrointestinal tract has been repeatedly demonstrated. Dopfer[9] reported a typical case with fatal outcome in a 2-year-old child, following repeated application of boric acid ointment to a burn measuring only 3 by 12 cm.

There have been conflicting reports in the literature concerning possible absorption of boric acid through unbroken skin. Kahlenberg[43, 44] conducted experiments in which he soaked the feet of volunteers in aqueous solutions of boric acid, and reported the presence of boric acid in the urine within a few minutes after immersion. More recently, Pfeiffer and co-workers[42] were unable to detect any absorption through unbroken skin surfaces, using a variety of topical applications over wide areas. The authors concluded that the urine in Kahlenberg's experiments must have been contaminated in some way with boric acid.

In order to clarify this point, we repeated Kahlenberg's experiment, using ten normal adult male volunteers. Control urine was first obtained from the subjects and the absence of boric acid confirmed by use of the turmeric paper test. (The turmeric paper was tested against boric acid solutions of known concentration, and was found to be sensitive to 1 part boric acid in 10,000 of water.) The volunteers then immersed their feet up to the ankles in foot baths containing a 4 per cent solution of boric acid. Urine specimens were collected at fifteen, thirty, and sixty minutes, and immersion was then discontinued. A final urine specimen was collected twenty-four hours later. In no case were we able to demonstrate the presence of boric acid in the urine.

This would seem to confirm Pfeiffer's contention that boric acid is not absorbed through the unbroken adult human skin. Recent studies by Johnstone and associates[49] indicate that the generally available borated baby powders constitute a hazard only when a considerable area of skin is denuded.

A statistical review of experimental lethal dosage of boric acid tends to be misleading, first, because death is an extreme criterion for the hazards of a poison, and, second, because the human recipient of the poison is not obliged by mathematical law to follow either the exact course of his fellows, or that of the laboratory animal. Nevertheless, it should be pointed out that the young infant is particularly susceptible to boric acid as a poison. In twenty fatal cases of this series, the approximate total amount of boric acid applied was known. It must be emphasized that such figures in no way suggest minimum lethal doses. How-

ever, in six members of the group under 2 weeks of age, reported by Young and collaborators,[37] the amount ingested accidentally was less than 3 Gm. in each instance.

There is ample evidence to indicate that the cases reported represent true poisoning rather than individual idiosyncrasy to the drug. This is particularly emphasized by the instances of "group poisoning" due to accidental inclusion of boric acid in infant feedings, and by Pfeiffer's studies in experimental poisoning.

The excretion of boric acid is mainly accomplished by the kidney; reports of clinical and experimental data indicate that between 85 and 100 per cent may be recovered from the urine. Small amounts are believed to be excreted in the feces, sweat, and saliva. Wiley[45] and Pfeiffer and co-workers[42] have demonstrated that both boric acid and borax are excreted unchanged.

Pfeiffer and associates[42] have also shown that the chief histopathological effects of experimental boric acid poisoning are found in the kidneys and brain. The kidneys showed glomerular damage, with debris in the glomerular spaces, and tubular degeneration, with coagulum in the lumina. The brain and spinal cord showed diffuse damage, with round cell infiltration, neuronophagia, and hyperchromatosis. Histological changes in the liver were minimal.

These authors also studied the metabolic effects of experimental boric acid poisoning. There was no detectable effect on the hematopoietic system. However, intravenous boric acid produced a consistent increase in urinary phosphorus excretion, and a questionable tendency to elevation of nonprotein nitrogen and serum potassium. Spectrographic studies of brain tissue indicated that phosphorus was probably displaced in brain tissue having a greater affinity for boron. It was also found that the addition of 0.25 per cent boric acid to the drinking water of immature rats had a definite inhibiting effect on growth.

DISCUSSION

The four cases presented illustrate the particular danger of poisoning in infants, following the application of boric acid preparations to skin eruptions. In the fourth case, the source of intoxication was the repeated application of a popular brand of baby powder, whose active ingredients are talc and boric acid.

There is indeed a strange paradox in the course and therapy of such infants. The usual story is that of a young infant who develops diarrhea with secondary excoriation of the buttocks. The boric acid is absorbed, and produces not only an increase in the excoriation, but also intensifies the diarrhea and vomiting.

The diagnosis of acute boric acid poisoning in infants is usually not difficult. The skin manifestations are fairly constant and typical. The erythema is usually intense and may extend over the entire body surface. The palms and soles are often particularly affected, and may give an appearance reminiscent of acrodynia, particularly when conjunctivitis and irritability are present.

The central nervous system signs may raise the possibility of meningitis, particularly in infancy. We have seen an unreported case in another hospital in a very young infant who was admitted with the clinical diagnosis of

meningitis, based on the findings of opisthotonos and a bulging tense fontanel. Lumbar puncture revealed normal cerebrospinal fluid; the diagnosis of boric acid poisoning was suspected and subsequently proved by an astute resident who noted the associated generalized erythema and excoriated buttocks.

The presence of boric acid in the urine or spinal fluid may be rapidly confirmed by the turmeric paper test. The solution to be tested is first strongly acidified with concentrated hydrochloric acid, and freshly prepared turmeric paper is then dipped in the solution and dried in air. The appearance of a pink to red color denotes the presence of boric acid. A rapid confirmatory test consists of placing a drop of concentrated ammonia water on the tested paper. A transient blue-black color confirms the presence of boric acid. Other acids produce a brown color on drying the paper, and a violet color with the confirmatory test. The test may be carried out in semiquantitative fashion by comparing the color reactions with those obtained from various known dilutions of boric acid solution.

We are convinced that many milder cases of boric acid intoxication go unrecognized. Many of us can look back on a number of infants with skin eruptions, irritability, some excoriation of the buttocks, and mild gastrointestinal upsets, and wonder about the possibility of boric acid absorption and toxicity. The reported cases are, for the most part, severe cases with a high mortality. This observation in itself suggests the likelihood that milder cases are frequently missed.

One cannot help but note the striking similarity of the manifestations of boric acid poisoning to those of the erythematous exfoliative dermatitis in the newborn and young infant, long known as Ritter's disease.

The similarity becomes still more suspicious when one recollects the details of the accepted newborn care of a few years ago, when "Ritter's disease" was more common than it is today. First, the conjunctivae were often bathed with boric acid solution. Then the umbilical cord was dusted liberally with boric acid powder. The infant's mouth was frequently swabbed with boric acid or borax and honey, and finally, the mother's nipples were cleansed with boric acid solution immediately prior to nursing. The newborn infant thus was provided with several avenues by which he might absorb toxic quantities of boric acid. It seems reasonable to suggest, then, that many infants, previously labelled with the vague diagnosis of Ritter's disease, may in fact have suffered from acute boric acid poisoning.

There is very little comment in the literature on the question of specific treatment for boric acid poisoning. Pfeiffer's group was able to increase the tolerance of experimental animals to the drug by the use of large doses of Ringer's solution and plasma intravenously, while mannitol, glucose, and glycerol were without antidotal effect. General supportive measures should consist of the treatment of shock with fresh plasma or whole blood, and the maintenance of a good urinary output with intravenous glucose solutions. We feel that excoriation of the buttocks is best treated by exposure to air and prevention of contamination with urine and feces.

Sedation may be necessary for the control of convulsions.

Boric acid has no bactericidal activity. It will inhibit the growth of certain organisms, but only temporarily.[47, 48] It adds nothing to the irrigating properties of water or physiological saline and yet its presence is still to be found in eye drops, ear drops, cleansing solutions, ointments, and baby powders. Its history in therapeutic usage is punctuated by an ever-increasing number of warnings against its toxicity and by testimonials to its lack of useful function. In pediatric practice, its use in the prevention and treatment of ammoniacal dermatitis is no longer defensible. We would recommend, therefore, that boron compounds in any form be withdrawn from use in medical therapy.

SUMMARY

1. Four cases of boric acid poisoning in young infants are presented. All four resulted from topical application of boric acid preparations to the buttocks. In one case a popular brand of baby powder was the source of toxicity.

2. One hundred nine cases of boric acid poisoning from the world literature are reviewed. The over-all mortality was 55 per cent, and the mortality in patients under 1 year of age was 70.2 per cent. The cases are analyzed with respect to age, route of intoxication, signs and symptoms, autopsy findings, and chemical analyses of organs, and these findings are correlated with the results of experimental boric acid intoxication.

3. The high degree of absorption of boric acid from broken skin surfaces and mucous membranes is emphasized. Absorption of boric acid through intact adult skin could not be demonstrated, confirming the findings of Pfeiffer and associates in this regard.

4. The particular sensitivity of young infants to the toxic effects of boric acid is noted, and the importance of recognizing milder cases of poisoning in this age group is stressed. The common clinical findings consist of erythematous skin eruptions followed by desquamation, diarrhea, and vomiting, and evidence of meningeal irritation. The presence of boric acid in the urine and spinal fluid is readily confirmed by use of the turmeric paper test.

5. We recommend that, since boric acid is a potentially dangerous drug, possessing only mild bacteriostatic activity, its use in any form of medical therapy, particularly as applied to infants and children, be discontinued.

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ADDENDUM

Since the above article was prepared for publication we have had the opportunity, through the courtesy of Dr. J. B. Scriver, of observing a fifth case of boric acid poisoning.

This was a 27-day-old infant who had had boric acid powder and a borated baby powder applied to a weeping umbilicus since the third day of life. The child had developed a marked erythema of the face and abdomen followed by desquamation. There were vomiting and convulsions. Boric acid in high concentrations was demonstrated in both the urine and spinal fluid. This case was of particular interest because the admission diagnosis by both a staff pediatrician and a dermatologist was Ritter's disease (see above). A history of exposure to boric acid was subsequently obtained.

The child has responded well to supportive therapy. Convulsions stopped twenty-four hours after admission, and boric acid had practically disappeared from the urine by the fifth hospital day. The infant is now gaining weight and appears to be recovering.

TRANSCUTANEOUS ABSORPTION OF BORIC ACID

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ABSORPTION of boric acid through broken skin and consequent fatalities have been reported frequently in the literature,[1, 2, 3, 4] yet the widespread use of this home remedy to dangerously absorptive skin areas still persists. The following report describes three cases of transcutaneous boric acid intoxication which were suspected on clinical grounds with subsequent confirmation by analysis of physiological fluids. The features of this syndrome are illustrated. The natural course of the illness and the method of supportive treatment are recorded. Interpretation of the clinical and laboratory data in these cases shows absorption reaching toxic levels to be a common clinical situation giving rise to dermatitis and gastrointestinal symptoms, the etiology of which heretofore has frequently gone unrecognized.

CASE REPORTS

CASE 1.—G. LaT., a 27-day-old white male infant, was admitted to The Roosevelt Hospital on July 18, 1950, with the diagnosis of gastroenteritis and a history of having vomited all feedings for three days. Previously the child had been well, and past medical history was noncontributory. The mother stated that the infant had vomited shortly after being fed on the evening of the third day prior to admission and continued to vomit all feedings during the night. His formula consisted of 10 ounces of evaporated milk, 20 ounces of water, and 2 tablespoons of Dextri-Maltose. The following day, two days before admission, the vomiting persisted and he retained but a little water, whereupon the family physician ordered a more dilute formula and additional water by mouth. That evening the mother noticed the appearance of erythematous patches over the fingers and toes, and later over the buttocks, nose, and tops of the ears. The pads of the toes and palmar aspects of the fingers were bright red. The child remained afebrile but began having loose stools and appeared drowsy. The day before admission, the rash had diffused over the entire body and three to four greenish colored bowel movements were reported. On the day of admission, the rash over the body cleared, but an excoriation of the buttocks appeared.

Physical examination revealed a well-nourished, moderately dehydrated infant weighing 8 pounds. Temperature was 98° F.; pulse 100, respirations 40 per minute. Considerable excoriation of skin over the scrotum was present. Fontanels were normal, but there was pronounced overriding of the bones in the right occipital region. Pupils and fundi were normal. Eyes, ears, and throat were not remarkable. Lungs were clear. Cardiac auscultation revealed no murmur nor arrhythmia. The liver was palpable 1½ fingerbreadths below the right costal margin. The abdomen was slightly distended. From time to time the child held his head in the opisthotonic position with his elbows flexed rigidly at his sides (Fig. 1). The neck was moderately stiff, there was definite hyperreflexia, and Babinski signs were present bilaterally. Neurological consultants felt that this clinical picture was not definitive of any particular intracranial lesion.

Laboratory data on admission was as follows: red blood cells 2,860,000; hemoglobin, 9.4 grams per 100 c.c.;

From the Pediatric Service of The Roosevelt Hospital.

J. PEDIAT.
VOL. 43, NO. 6,
DEC., 1953

Fig. 1.

Fig. 2.

Figs. 1 and 2.—Case 1. Boric acid intoxication in a 27-day-old infant, showing severe perineal erythematous excoriation, complete desquamation of the ears, hands, and feet, periorbital edema, flexed forearms and clenched hands with opisthotonus.

white blood cells 10,800 with polymorphonuclear neutrophils 22 per cent, nonsegmented forms 67 per cent, and lymphocytes 11 per cent. Urinalysis was normal with specific gravity of 1.020. Blood calcium was 9.7 mg. per 100 c.c.; phosphorus 4.2 mg. per 100 c.c.; chloride 85 meq. per liter; CO_2 21 meq. per liter. Urea nitrogen was 11.8 mg. per 100 c.c. Total protein was 5.08 with albumin of 3.58 and globulin 1.58 grams per 100 c.c. Cephalin flocculation and thymol turbidity were 1+. Spinal fluid: normal pressure, 5 lymphocytes per cubic millimeter, protein 19 mg. per 100 c.c., and sugar 61 mg. per 100 c.c.

Films of the skull and chest were negative. Tuberculin 1:10,000 and serology tests were negative. Nose culture revealed heavy growth hemolytic *Staphylococcus aureus*, and throat culture, heavy growth of pneumococcus, moderate growth of hemolytic staphylococcus, and light growth of beta hemolytic streptococcus. Blood and cerebrospinal fluid cultures were negative. Repeated stool cultures were negative for enteric pathogens.

Course and Treatment.—The child continued to vomit and have diarrhea after admission and was sustained with intravenous saline, glucose, whole blood, and the administration of oxygen. There was no clinical evidence of tetany. On the second day the opisthotonos was more marked although hydration was good. The increased tone of the extremities, especially in the arms, became more pronounced, and there were now intermittent coarse tremors with persistent hyperactive reflexes. The child's condition remained critical. He experienced repeated convulsions. His respirations progressively slowed, and because of a great deal of respiratory difficulty, external stimulation was necessary. At this time the skin began to peel over the ears, head, abdomen, legs, and especially the finger tips. The left eyelids were erythematous and slightly swollen. The excoriations of the scrotum by now had become severe. (See Fig. 2.) The entire penis, scrotum, and contiguous areas of the groin were markedly swollen and crimson in color. Venous blood drawn at this time was a bright red, and the urine on several occasions showed a definite yellowish-green fluorescence. The white cell count had dropped to 5,440 with 62 per cent neutrophils loaded with eosinophilic toxic granules, 30 per cent lymphocytes, 5 per cent monocytes, and 3 per cent eosinophils.

In view of the close similarity of this case to those of boric acid poisoning reported in the literature,[2, 3, 5, 6, 7] the mother was questioned more closely. It was established that a commercial borated talc followed by boric acid powder had been used on a diaper rash (perianal dermatitis) for seven days prior to admission. Samples of blood and urine sent to the office of the Chief Medical Examiner of the City of New York on the second hospital day showed 5.0 mg. per 100 c.c. of boric acid in the blood and 28 mg. per 100 c.c. in the urine.[8]

Supportive treatment consisted of the intravenous administration of fluids and whole blood to guard against the possibility of circulatory collapse and renal shutdown. To enhance further the excretion of boric acid, fluid intake was elevated to 200 c.c. per kilogram of body weight, and 0.5 c.c. of a mercurial diuretic was given intramuscularly. Penicillin and streptomycin were given prophylactically because bronchopneumonia has been listed as a cause of death in some of the cases reported.[4, 9] Caffeine improved the respiratory rate considerably during periods of irregular breathing and intervals of apnea. Convulsive seizures and spasms were lessened to a great degree with sodium phenobarbital sedation in the amount of 0.015 Gm. intravenously every six hours. Skin therapy was restricted to the generalized application of olive and mineral oils, with small amounts of Lassar's zinc paste to the scrotum and buttocks.

By the fifth hospital day the child appeared much better and was taking fluids by mouth. The opisthotonos was less, but some fibrillary twitchings of the arms and legs were noticed at this time. Hydration appeared good and the heart sounds were normal. By the eighth hospital day the opisthotonos had disappeared, and all reflexes were within normal limits. There was no further desquamation. The skin of the hands and feet appeared normal. The scrotum and surrounding areas were dry and almost healed, and the edema of the penis had subsided. The liver was still palpable 1½ fingerbreadths below the costal margin. The remainder of the physical examination was negative.

Prognosis appeared favorable approximately eight days after admission and the child improved daily. He was discharged home on the eighteenth hospital day, at which time he was free from symptoms and was taking his feedings well. Blood studies shortly prior to discharge were normal.

CASE 2.—G. L., a 4-month-old premature white female infant, was admitted Nov. 24, 1952, with diarrhea and dehydration. She had made excellent progress while hospitalized for the first two months of life after a premature birth as one of twins at seven months' gestation, weighing 2 pounds 4 ounces. One month prior to entry she was seen at another clinic for a dermatitis for which no treatment was considered necessary. The mother, however, applied boric acid ointment for one week to the involved area, mainly the diaper region. When no improvement followed, she bathed the child in boric acid solution and dusted her skin with boric acid crystals intermittently during the following two weeks. The diaper rash became worse and had seemingly spread to other parts of the body, so that she discontinued the use of boric acid containing compounds during the week prior to admission. Four days before admission the baby began to have six to seven loose stools daily. The formula of 14 ounces of evaporated milk, 17 ounces of water, and 3 tablespoons of Karo was taken fairly well without vomiting other than occasional regurgitation. The day before entry the mother thought the infant was hoarse and had a sore throat which she treated with Argyrol and Vicks ointment. When the child began having difficulty breathing, the mother became alarmed and brought her to the Emergency Ward.

Physical examination showed an emaciated, cyanotic, dehydrated female infant in considerable respiratory distress breathing 70 times a minute. Temperature was 98° F., pulse 140. There was an extensive scaling with drying of the exfoliated portions of the epidermis and reddening of the underlying dermis involving the neck, scalp, posterior surface of the ears (Figs. 3 and 5), and most severely the groins, thighs, vulva, perineum, and buttocks (Figs. 5, 6, and 7). There were small patches of fine exfoliation over the chest, abdomen, fingers, and toes. The lips were dry and cracked (Fig. 7). The fontanel was flat. Otherwise the eyes, ears, nose, and throat were unremarkable. Her cry was aphonic and she lay in an opisthotonic position (Figs. 5 and 7) with rigid limbs and flexed elbows, twitching from time to time. The reflexes were hypoactive. Heart, lungs, and abdomen were negative on admission. She was passing very soft, yellow stools. Her weight was 7 pounds.

Laboratory data on admission showed a red cell count of 4,070,000; hemoglobin, 9.5 grams per 100 c.c., with anisocytosis, hypochromia, polychromasia, and 5 normoblasts per 100 white cells. White cell count was 32,200 with neutrophils 34 per cent, nonsegmented forms 12 per cent, lymphocytes 46 per cent, monocytes 7 per cent, and eosinophils 1 per cent. Blood chlorides were 123 meq. per liter, serum potassium 5.4 meq. per

J. PEDIAT.
VOL. 43, No. 6,
DEC., 1953

Fig. 3.

Fig. 4.

Fig. 3.—Case 2. Shows the female twin with a clinical picture similar to that of Case 1. This infant expired. Age: 4 months.

Fig. 4.—Case 3. Shows the male twin with less severe transcutaneous boric acid intoxication, in that there were few central nervous system manifestations but severe erythema and excoriation.

Fig. 5

Fig. 7

Fig. 5.

Fig. 6.

Fig. 7.

Fig. 8.

Figs. 5-7.—Case 2. Additional photographs showing the severe central nervous system involvement and cutaneous reaction.

Fig. 8.—Case 3. Shows extensive skin involvement and axillary intertrigo, with minimal neurological signs.

liter, and CO_2 5.4 meq. per liter. Kline and Mazzini tests were negative. X-ray of the chest was negative.

Course and Treatment.—Rehydration was begun with parenteral saline, dextrose, lactate, whole blood, and small amounts of calcium gluconate. On the second hospital day the CO_2 rose to 22.8 meq. per liter, the chlorides were 116.6 meq. per liter, and the sodium 150 meq. per liter. Bacitracin ointment was applied to the skin lesions. After rehydration the boric acid level of the blood[8] was 2.2 mg. per 100 c.c. The cerebrospinal fluid was clear and showed no increase in pressure, protein of 28 mg. per 100 c.c., sugar 105 mg. per 100 c.c., and chlorides 121 meq. per liter, with 62 red cells and 19 white cells per cubic millimeter. Cerebrospinal fluid culture was negative. It contained 5 mg. of boric acid[25] per 100 c.c. The urine output which was very low on admission gradually became adequate. It was yellow, cloudy, contained 1+ albumin, occasional white cells and many bacteria. Three stools were negative for enteric pathogens.

The child's condition remained critical although the diarrhea stopped. The cyanosis continued in spite of oxygen. The opisthotonus persisted. She developed râles in the lower left lung field with decreased breath sounds and peribronchial infiltration by x-ray. The temperature persisted at subnormal levels. Dyspnea continued and at one point reached 85 per minute. Intravenous terramycin was added. She took nothing by mouth and her parenteral fluids were amplified by gastric tube feedings, through which ammonium chloride was administered when the chlorides fell to 70 meq. per liter on the fifth hospital day. On the sixth day the child began to have Cheyne-Stokes respiration and subsequently stopped breathing.

The Medical Examiner of the City of New York certified that the cause of death was "toxemia and exfoliative dermatitis following topical application of powdered boric acid in the form of dusting powder for the treatment of an acute eczematous dermatitis." Tissue analysis revealed: 21 mg. boric acid per 100 grams of kidney, 10 mg. per 100 grams of liver, 1.2 mg. per 100 grams of brain, and 1.1 mg. per 100 grams of muscle. Autopsy also showed atelectasis of the left lung; cerebral, hepatic and renal congestion; vacuolar and granular degeneration of the convoluted tubules, and pancreatic vacuolar acinar lesions corresponding to that described by Fisher[9] in acute boric acid intoxication.

CASE 3.—S. L., a 4-month-old premature white male infant, twin brother of Case 2, was admitted Nov. 25, 1952, for evaluation of dermatitis treated with boric acid according to same regimen as his sister. Birth weight was 2 pounds 10 ounces. He had intermittent diarrhea for one month prior to admission, and the mother brought the child to hospital after he had vomited feeding. The formula was the same as that for his twin. There was no respiratory distress.

Physical examination showed a moderately dehydrated male with slightly depressed fontanel. Temperature was 99° F., pulse 110, respirations 30 per minute. There was a shiny red excoriation over the lower abdomen, perineum, buttocks, groins, and intertriginous areas of the neck and axillae. At the periphery of these lesions there was moderate exfoliation (Figs. 4 and 8). Other than moderate irritability, the child was negative neurologically, and there were no other abnormal physical findings. Weight was 7½ pounds.

Laboratory data showed a red cell count of 3,300,000 hemoglobin, 10 grams per 100 c.c., and hypochromia. White cell count was 10,600 with 11 per cent neutrophils, 4 per cent nonsegmented forms, 81 per cent lymphocytes, and 4 per cent monocytes. Urinalysis showed 1+ albumin and many bacteria. Kline and Mazzini

tests were negative. Admission CO_2 was 17.2 meq. per liter. Chlorides, potassium, sodium, and CO_2 were normal on the sixth day. Chest film was negative. Tuberculin 1:10,000 was negative. On admission, boric acid blood level[8] was 1.88 mg. per 100 c.c. and urine boric acid level was 3 mg. per 100 c.c.

A watery diarrhea developed shortly after admission but promptly remitted. He took oral feedings well and required parenteral fluids for only the first day. The hyperirritability lasted about one week. The skin lesions cleared very slowly on potassium permanganate soaks and calamine lotion. The possibility of moniliasis of the skin was raised, but scrapings grew heavy staphylococcus and anaerobic streptococcus. Penicillin was given prophylactically and reinstituted when the child developed a bronchitis in the third hospital week. Otherwise recovery was uneventful.

COMMENT

In recent years overwhelming evidence proving the toxicity of boric acid has been gathered,[10, 11, 12] and wholesale fatalities have occurred following the accidental ingestion of this material.[13, 14, 15] But it is the widespread prevalence of its use as a topical agent which brings out the most important clinical complication of boric acid therapy: its transcutaneous absorption.

Because it is generally believed that boric acid is not absorbed from an intact skin surface in significant amounts, the false impression prevails that it is a harmless agent. However, application to an inflamed or injured area immediately introduces a serious problem, for cumulative absorption in toxic quantities may occur. Although deaths have been reported in adults, they usually recover, especially if the skin lesion heals promptly with concomitant early withdrawal of boric applications.[16] This is again misleading, for what has been generally accepted in treatment of adult skin disorders takes on lethal proportions in infants.[17]

In these cases boric acid powder, a commonly used borated talc, and boric acid ointment were responsible for the intoxication. Percutaneous absorption to cause intoxication appears to be made possible by conversion of the boric acid dry particles to boric acid solution when collection of urine occurs about the diaper rash region to which the boric-acid-containing powders were applied. Intoxication from borated tales* and 10 per cent borated ointments[2, 11, 18] have previously been reported. Fisher,[9] in his study of the unexpected deaths of infants in Baltimore in the past two years, where the usual history was that of development of a diaper rash in an infant under one year of age and treatment of the dermatitis by repeated application of boric acid in talc or starch mixtures, concludes that there is real danger of poisoning by boric acid absorption through areas of dermatitis. This agrees with a nonfatal poisoning reported by Brooke[19] wherein a dusting powder of 25 per cent boric acid in a starch base was stated to be the direct cause for the intoxication.

It should be re-emphasized that the admission diagnosis in the first two cases was gastroenteritis. We are in agreement with others[9] in stressing the probability that many more cases of boric acid poisoning go undiagnosed and are considered erroneously to be feeding problems or other types of gastrointestinal disorders.[20, 21, 22, 23]

*Chemical analyses disclose that many commercial baby powders contain 5 per cent boric acid, others in excess of 10 per cent.

Should death occur, it is likely to be ascribed to a terminal complication, such as pneumonia or meningitis,[9, 11] rather than to the real cause of death: boric acid absorption. In the absence of a history of chemical ingestion, the usual autopsy does not include analyses for intoxicating agents.

The ease with which morbidity secondary to boric acid may go unrecognized is strikingly illustrated in Case 3 (Figs. 4 and 8). The surviving male twin, with as great an *area* of skin involvement as that of his sister who died, had apparently absorbed less boric acid under similar home medication—or had tolerated it better. Since the mother had discontinued boric acid application, he would have continued to improve without therapy. His intoxication would have gone unrecognized had the mother not been alerted by the fact that his twin sister became seriously ill and was admitted to the hospital the previous day.

Calculations.—The potential cumulative transcutaneous absorption of boric acid from solution resulting from 5 per cent borated talc in contact with urine may be calculated, using the following representative facts: (1) The average area covered by dusting powder during a diaper change of a 3-month-old infant, for example, is 725 sq. cm. (2) The average quantity of dusting powder used during a diaper change is 5 Gm., or 50 Gm. for ten diaper changes. (3) The excretion of urine for infants during a twenty-four-hour period is 600 to 1,000 c.c.

Five grams of a dusting powder containing 5 per cent boric acid has actually 0.25 Gm. of boric acid. Ten diaper changes therefore would effect a daily deposit of 2.5 Gm. upon the whole gluteal territory. The N. F. formula for a saturated solution of boric acid is 50 Gm. in 1,000 c.c., or a 5 per cent solution (weight/volume). With a daily excretion of 600 to 1,000 c.c. of urine in twenty-four hours, there is available sufficient liquid to dissolve all of the boric acid present in ten applications of the dusting powder (borated 5 per cent).

TABLE I

AREA INVOLVED		10 SQ. CM.			100 SQ. CM.			300 SQ. CM.		
PERCENTAGE ABSORPTION		100% (GM.)	25% (GM.)	1% (GM.)	100% (GM.)	25% (GM.)	1% (GM.)	100% (GM.)	25% (GM.)	1% (GM.)
Number of days of 10 daily applications	1	0.0345	0.0086	0.00035	0.345	0.086	0.0035	1.035	0.258	0.0104
	2	0.0690	0.0172	0.00069	0.0690	0.172	0.0069	2.070	0.516	0.0207
	5	0.1725	0.0431	0.00173	1.725	0.431	0.0173	5.172	1.293	0.0516
	10	0.3450	0.0862	0.00345	3.450	0.862	0.0345	10.350	2.586	0.1035

Based upon an availability for absorption of 2.5 Gm. of boric acid in ten dusting applications within twenty-four hours over a 725 sq. cm. area, Table I shows the quantities available for absorption by excoriated areas of 10, 100, and 300 sq. cm.

In Case 1 the boric acid blood level was determined by chemical analysis to be 5.0 mg. per 100 c.c. of blood. The total amount of boric acid in the circulation would thus be 17.5 mg., assuming the blood volume to be approximately 350 c.c. As this child was moribund it would appear that this figure is close to the fatal blood level.

Although there are no accurate figures on the rate of variable diffusion of boric acid into the various

tissues of the body, it is known that it is readily absorbed through the injured skin. From the above calculations it may be seen that relatively large quantities of pure boric acid are available for absorption even when a 5 per cent dusting powder is used. The lethal amount of 17.5 mg. is reached in only five days over the relatively small area of 100 sq. cm. if only 1 per cent of the *available* boric acid accumulates in the blood stream.

In conclusion, in view of the insidious nature of boric acid poisoning and the facility of its transcutaneous absorption, it seems likely that further morbidity will result unless its use is discontinued in favor of other more efficient and safe antiseptics.[24, 25, 26, 27]

SUMMARY

1. Three case of transcutaneous absorption of boric acid in infants treated for diaper rash are reported. One child died. The diagnoses were made on clinical grounds and corroborated by laboratory analysis of blood, urine, and cerebrospinal fluid.

2. Borated dusting powders, widely sold commercially, are shown to be capable of forming saturated solutions in common clinical situations with possible resultant absorption of toxic quantities through broken skin.

3. The use of boric acid should be discontinued in favor of safe and efficient antibacterial agents that are now available.

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PERTUSSIS IMMUNIZATION IN CHILDREN WITH CONVULSIVE DISORDERS

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IN SWEDEN many misunderstandings and false interpretations still adhere to the diagnosis of epilepsy, which makes the work with the seizured patients a thrilling task. Year after year it is, however, brightening a little. In recent years the school-physician-in-chief and the administrative school authorities in Sweden have taken a very sympathetic view toward the educational possibilities for children with convulsive disorders.

Recently a problem has come up about these children which is of considerable importance, and which needs to be discussed. In 1948 Byers and Moll published an article on encephalopathies following prophylactic pertussis vaccine. These authors reviewed earlier articles on the subject and discussed briefly the published cases, where convulsions and other neurological disorders had followed at varying intervals after the injection of pertussis vaccine. They added 15 of their own cases from the records of the Children's and Infants' Hospital in Boston during a ten-year period. They also found about twice as many children during this period of time, suffering from an encephalopathy, complicating pertussis itself.

This publication led Toomey to make a nationwide survey of pediatricians in the United States, through which he disclosed 38 cases of neurotoxic reaction to pertussis vaccine from among scores of thousands of immunizations. Toomey closes his presentation of this material with a number of recommendations, among which I quote the following:

> No child should receive injections of pertussis vaccine in large amounts (40 billion organisms, double strength) who has a) any family history of convulsions, b) a present history of convulsions or c) illness of any kind, especially if it pertains in any way to the central nervous system.

This statement has subsequently been more or less generally accepted, and these recommendations have later also been applied to the triple vaccine, where pertussis vaccine is one of the three components. Every year new reports are published of complications after pertussis or combined immunizations, and usually only occasional cases are reported. In some of them Toomey's recommendations are even strengthened. For example in 1953 Sutherland stated: "A family or personal history of any neurological disease such as epilepsy should contraindicate anti-pertussis inoculation." Miller (1951) is a little more liberal. He says: "Children with known cerebral damage should not receive H. pertussis vaccine or diphtheria toxoid. Children with histories of febrile convulsions should be inoculated first with a fractional dose."

Without doubt there is evidence from many places which shows clearly that the immunizations mentioned entail some risk of neurological complications. This risk is of course greater in children with any type of disorder in-

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volving the central nervous system. If this risk of encephalopathy from vaccination was the only consideration, I would agree with those authors who advise against immunization for pertussis in children predisposed to convulsive disorders.

In my opinion it is, however, necessary to look upon this question from another point of view. During my work with seizured children in Stockholm, which started with work in Dr. Lennox's clinic in Boston in 1946, I have repeatedly found that the convulsive disorder temporarily or permanently has worsened during clinical pertussis. I will refer to two typical cases.

A pair of identical twins (EEG nr KLB 65 and 66) started without any obvious cause to have petit mal attacks at the age of 5 years, 9 months and 5 years, 11 months. Their disease developed in an almost identical way, and at the age of about 8 years they each had four to five observed daily attacks. On Tridione treatment they both became almost seizure-free. At the age of 8 years, 6 months they were both without any Tridione for about three weeks. During this period each had ten to twenty observed daily attacks. When placed on Tridione again, they both improved. When the boys were about 9 years old, one of them contracted a severe pertussis, with a whooping period of about five weeks. During the illness his petit mal became markedly worse, and from this time the twins have developed in quite different ways. One of them was able to reduce his Tridione dosage gradually, and he has had no treatment for more than two years. He is seizure-free and his electroencephalogram (EEG nr KLB 66/3693) shows no wave and spike periods, even with hyperventilation and simple photic stimulation. The other one has continued to be worse after his pertussis, and he has now many daily petit mal seizures, in spite of series of trials with different drugs. His electroencephalogram (EEG nr KLB 65/6558) shows clinical and subclinical petit mal periods, with typical wave and spike complexes.

We thus have a pair of identical twins with petit mal, where the disease proceeded in exactly the same way until a point where one of the boys contracted a severe pertussis. During the course he became worse from a seizure standpoint. Subsequently, the pertussis-free twin recovered from his petit mal while in the other one a deteriorated seizure situation continued five years after the pertussis.

In another case (EEG nr KLB 1896) a 7-year-old boy, with occasional grand mal attacks after toxoplasmosis at the age of 7 months, contracted pertussis. Following the disease his grand mal attacks came at much shorter intervals than before. Also in this case the impairment had remained. Other cases in my clinical material show a similar course, and to date I have collected 10 cases where seizured children of varying ages have shown a temporary or permanent exacerbation of their convulsive disease after clinical pertussis.

In view of the foregoing considerations we must very carefully weigh, on one side, the risks of encephalopathy, involved by the immunizations in question, and, on the other side, the risks of the seizured child's contracting pertussis and so possibly experiencing an exacerbation of his convulsive disorder. I have considered the last-mentioned

risk to be the major one. I have therefore started to vaccinate seizured children with pertussis or triple vaccine. Together with Dr. Thorsell-Lagergren I have now immunized altogether 47 children with different types of convulsive disorders, 45 with pertussis vaccine and two with triple vaccine. To this can be added 30 Mongols in an institution, all pertussis-immunized. All children but three have received the vaccine in full dose. In a few cases the intervals between the injections were increased above the usual. Two of the children have received booster doses at one- to two-year intervals. The age of the children immunized has been in 6 cases under 2 years, in 5 cases between 2 and 3 years, in 15 between 3 and 5 years, and in 21 over 5 years of age.

All vaccinations but one have been without complication. The children have had the usual local pain and some swelling, and in some children there has been a moderate rise in the temperature. No sudden high temperature has been noted, and no convulsions were precipitated.

In one single case (EEG nr KLB 3992) the course was not uncomplicated. A girl was immunized against smallpox at the age of 10 months. Thirteen days later she started to have convulsions, and during the next two months she had six major fits. At this time there was an epidemic of pertussis in the vicinity. She had one pertussis vaccine injection, without any complication. After the second injection she had major fits on the three succeeding days. She was put on phenobarbital treatment and had no more attacks and escaped clinical pertussis. Now, two years later, she presents delayed speech development, as does her brother, two years older than she.

In conclusion I would like to recommend pertussis or triple vaccination for children with convulsive disorders. I base my recommendation partly upon personally observed cases, in which clinical pertussis has made a pre-existent convulsive disorder worse, partly upon our experience with such vaccinations in a series of seizured children, in whom the immunization procedure has given no real trouble. In my opinion the risk of a seizured child's contracting pertussis and eventually developing complications is more pronounced than the risk involved in the immunization procedure itself. It is, however, appropriate to follow the children carefully during the immunization and to interrupt the procedure at any signs of complication, especially if a convulsion occurs in connection with the immunization. If high fever follows the injection, phenobarbital could be given, or pre-existent anticonvulsive medication could temporarily be increased.

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SICKLE-CELL ANEMIA CRISIS

Report on Seven Patients Treated With Priscoline

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SICKLE-CELL anemia is a disorder in which the clinical manifestations are so pleomorphic[1] as to simulate many other well-known disease entities. Rheumatic fever,[2] poliomyelitis,[3] encephalitis,[4] and acute abdominal surgical crises[5] are among the better known conditions from which sickle-cell anemia must be differentiated.

In the course of treating the pain of vasospastic origin in poliomyelitis with Priscoline,[6, 7, 8, 9] a striking similarity to the pain of sicklemic crisis was noted. The drug was therefore given a clinical trial in seven patients suffering from the disease. Problems in diagnosis were in some cases quickly resolved by the use of Priscoline. It was therefore thought worthy to report the use of Priscoline not only for its value in the relief of the pain of sicklemic crisis but also for its use as a diagnostic tool in the differential diagnosis of sickle-cell disease. Representative case reports are as follows:

CASE REPORTS

CASE 1.—*Sickle-Cell Anemia Crisis Simulating Rheumatic Fever.*

E. B., a Negro boy aged 5 years, was admitted to the Kingston Avenue Hospital on Feb. 28, 1949, with a one-day history of fever, coryza, and sore throat. Physical examination revealed a well-developed, and well-nourished child with a temperature of 105° F., pulse 120, and respirations 32. The nasal mucosa was congested and there was a profuse mucoid discharge. The tonsils were enlarged, red, and covered with a gray membrane. The cervical lymph nodes were enlarged and tender. The heart was not enlarged but a soft systolic murmur was heard best at the third left intercostal space. The skin revealed a diffuse scarlatiniform eruption. An admission diagnosis of scarlet fever was made for which the child was treated. Seven days after admission the child became afebrile and the rash faded. The skin about the abdomen and fingernails began to desquamate. The soft systolic murmur found on admission was now accentuated. The child complained bitterly of pain about both knees and he favored a fixed side position with the knees drawn to his chest. The pain in the joints was aggravated by motion. The abdomen was soft but tender. Because the child was in the convalescent period following scarlet fever and the cardiac murmur found on admission was now accentuated, and because of the appearance of joint pain, rheumatic fever was suspected. During a routine blood count sickling of cells was noted. The hemogram showed 7 grams of hemoglobin; erythrocytes 2,480,000 and leukocytes 14,000 with 62 per cent polymorphonuclear leukocytes, 32 per cent lymphocytes, and 6 per cent monocytes. The erythrocytes were hypochromic and showed anisocytosis and poikilocytosis. Bone marrow study showed moderate erythroblastosis and normal leukogenesis and platelet formation. Roentgen-ray examination of the bones was negative for pathology

From the services of the Kingston Avenue Hospital and the University Division of the Kings County Hospital Center.

associated with sickle-cell anemia. Twenty-five milligrams of Priscoline was given intravenously on March 7 followed by 50 mg. of the elixir of Priscoline every three hours during the day and every four hours during the night. Following the intravenous administration of the drug there was almost instantaneous relief from the pain in the extremities. That evening the child was active and offered no resistance to examination. The following morning Priscoline was discontinued. Late that afternoon pain in the lower extremities and in the right arm reappeared. The child was up several times during the night complaining of pain and when examined the next morning tenderness in the extremities was noted similar to that experienced before therapy was begun. Treatment with Priscoline was reinstituted and continued for eight days with excellent results. The child was then transferred to the sending hospital for treatment of the sickle-cell anemia.

CASE 2.—*Sickle-Cell Anemia Crisis Simulating Poliomyelitis.*

E. I., aged 3 years, developed a sore throat and articular pain on Sept. 4, 1952. Two days later the temperature rose to 103° F. and a physician at a local hospital clinic administered penicillin intramuscularly but refused to admit the child to the hospital. The following day because of severe pain in the lower extremities, the child was brought back to the clinic from which he was admitted to the pediatrics ward. Poliomyelitis was suspected and a lumbar tap was done. The spinal fluid was clear and there were 116 lymphocytes per cubic centimeter. The child was then transferred to the contagious division of the Kingston Avenue Hospital.

Physical examination on admission revealed a well-developed, well-nourished Negro child who was apprehensive and uncooperative. His throat was congested and the tonsils hypertrophied; the tongue was dry and coated. Every part of his body seemed to be painful and the child cried whenever he was touched. His head was held in a fixed position and could not be moved in any direction. It could not be determined whether the resistance was voluntary or involuntary. A soft systolic murmur was heard in the left third intercostal space near the nipple line. The reflexes of the upper extremities were hyperactive. The abdominal reflexes were sluggish. The tendon reflexes of the lower extremities could not be elicited. The back muscles were in spasm and stiff. Bilateral Kernig and Brudzinski signs were noted. The child at times was semistuporous and he would not talk; he stared into space vacantly. At other times he was irritable and fretful. The latter was attributed to a behavior problem and accordingly another Negro child with similar spinal fluid findings was placed in the same room in an attempt to cope with the problem. Spinal fluid study was repeated and at this time no cells were found. Total protein was 15 mg. per cent and sugar 92 mg. per cent. A blood count showed hemoglobin of 6.25 grams per cent; erythrocytes 2,300,000; leukocytes 18,600 with a differential of 57 per cent polymorphonuclear leukocytes, 32 per cent lymphocytes, 5 per cent eosinophils, and 1 per cent basophils. The red blood cells were hypochromic and microcytic. A sickle-cell preparation was positive. The diagnosis was accordingly changed to the crisis of sickle-cell anemia and the second child was removed from the room. Twelve and a half milligrams of injectible Priscoline was immediately given intravenously. The pain in the extremities subsided, the child sat up, walked, and ceased to be a behavior problem. He received two transfusions, each of 250 c.c. of whole blood given two days apart. The neuromuscular symptoms rapidly disappeared and when discharged the hemoglobin was 11.5 grams per cent and red blood count 4,200,000.

CASE 3.—*Sickle-Cell Anemia Crisis Simulating Dysentery.*

D. J., a 10-year-old Negro female, was admitted to the University Division of the Kings County Hospital Center on Nov. 8, 1952. Her chief complaint was severe abdominal pain and diarrhea of two days' duration. She also stated that severe backache had developed two hours before admission.

Physical examination found the child to be in moderate distress. The heart was enlarged to the left and there was a coarse systolic murmur over the apex transmitted to the mid-axillary line. There was moderate abdominal tenderness and many borborygmi. The lumbosacral areas were extremely painful. All superficial and deep reflexes were hyperactive. The temperature was 100° F., pulse 120, and blood pressure 110/80. A diagnosis of dysentery was tentatively made but the hemogram revealed the true cause of the clinical picture. The hemoglobin was 7.2 grams per cent; erythrocytes 2,250,000; leukocytes 26,500. The differential showed 63 per cent polymorphonuclear leukocytes, 18 per cent lymphocytes, 7 per cent monocytes, and 1 per cent eosinophils. Many sickled cells and target cells were seen in the peripheral blood smears and a sickle-cell preparation was positive. Urobilinogen was positive in a 1:8 dilution.

The child was given 25 mg. of Priscoline intramuscularly. The abdominal pain subsided in two to three minutes but the low back pain persisted. The injections were repeated twice at four-hour intervals with no appreciable relief of the back pain. She then was given codeine and aspirin and a transfusion of 500 c.c. of blood. In a few hours she was completely asymptomatic and was discharged three days after admission. It should be noted that while the Priscoline effectively relieved the abdominal complaints, there was no effect on the low back pain.

CASE 4.—*Sickle-Cell Anemia Crisis Simulating Acute Encephalitis.*

B. E., an 11-year-old Negro female, was admitted to the University Division of the Kings County Hospital Center with a history of convulsions which lasted two hours prior to admission. The child was known to have had sickle-cell anemia since the age of 4 years and had been previously treated with blood transfusions. Two days prior to admission she had become progressively pallid and lethargic, and she vomited several times. The day before admission she complained of pain in both knees and had vomited all alimentation. Two hours before admission she suffered generalized clonic convulsions and was brought to the hospital.

Physical examination revealed a fairly well-developed mulatto female who was stuporous and thrashing about. The skin was pale with an icteric tinge. The temperature was 102° F., pulse 140, and blood pressure 106/70. Respirations were shallow. The pupils were round, regular, and reacted to light but not accommodation. The tonsils were very large and red. The lungs were clear. The heart showed sinus tachycardia. The spleen was firm and palpable at the level of the iliac crest. The liver was not felt. Neurological examination elicited no abnormal reflexes. The neck was supple.

The hemoglobin was 7 grams per cent and many sickled cells were seen in smear preparations. Spinal tap showed clear fluid under normal pressure. No cells were seen and cultures and smears failed to reveal organisms. The total protein was 16.5 mg. per cent, chlorides 745 mg. per cent, and sugar 95 mg. per cent.

On the morning of the second hospital day the child had three convulsions which were controlled by Sodium Luminal. These recurred in the evening and were controlled by the same method. At this time it was noted that the right leg was paretic. The child was stuporous and twitch-

ings of the left side of the face were noted. The temperature rose from 102° to 104° F. She was placed in an oxygen tent and intravenous glucose given. On the third hospital day she was still stuporous and the temperature fluctuated between 102° and 103.2° F. The eyes were wandering and the extremities moved aimlessly. Deep tendon reflexes were absent. During the morning of the third hospital day the child was given 10 mg. of Priscoline intravenously through the tubing of the glucose solution. One and one-half minutes following the injection, the patient became perceptibly flushed. Her pulse rose to 160 but promptly returned to the previous level of 120. The blood pressure remained unchanged. The child was given 10 mg. of Priscoline every four hours, and after the third injection she awoke somewhat disoriented but smiling, and took fluids by mouth. The next morning she appeared drowsy but well oriented and responsive. The temperature varied between 100° and 101° F. Some suggestion of weakness on the left side persisted. She continued to improve, and on the sixth hospital day or three days after Priscoline therapy was begun, the child was completely free of clinical signs of the hemolytic episode. She was discharged from the hospital on the fifteenth hospital day.

CASE 5.—*Sickle-Cell Anemia Crisis Simulating an Acute Abdominal Emergency.*

There were three patients who fell into this group but only one case history will be given in detail.

B. E., a 6-year-old Negro male known to have sickle-cell disease, was admitted to the University Division of the Kings County Hospital Center, July 10, 1952, with a chief complaint of sudden onset of severe abdominal pain.

Physical examination showed the child to be in acute distress. The legs were drawn up to the chest. The abdomen was soft but painful. The heart was not enlarged but a soft systolic murmur was heard. The temperature was 99° F., pulse 120, and respiration 30. The hemoglobin was 7 grams per cent with many sickled cells in the direct smear. Soon after admission 10 mg. of Priscoline was given intramuscularly with relief of the abdominal pain, and the child fell asleep. Three hours later he awoke complaining of recurrent abdominal pain and again was given Priscoline in increased dosage of 25 mg. Priscoline had to be administered at three-hour intervals to keep the child free of pain and he received five injections in all. On the second hospital day the hemoglobin was 7.5 grams per cent, and the white blood count 20,000, with 56 per cent polymorphonuclear leukocytes, 32 per cent lymphocytes, and 2 per cent eosinophils. A transfusion of 250 c.c. of blood was given. On the third hospital day he had a recurrence of abdominal pain which was relieved with a single dose of Priscoline. He was discharged two days later clinically well with a hemoglobin of 9 grams per cent.

The child was readmitted to the hospital July 31, 1952, with a recurrence of severe abdominal pain. At this time the abdomen showed generalized voluntary splinting and tenderness throughout. The hemoglobin was 8 grams per cent, and the white blood count 28,500 with 92 per cent polymorphonuclear leukocytes, 6 per cent lymphocytes, and 2 per cent monocytes. There was marked poikilocytosis and sickling of the erythrocytes. Twenty-five milligrams of Priscoline was given intramuscularly, and the child fell asleep two minutes after the injection. He required the drug at three-hour intervals to keep him symptom-free, and after the third day of hospitalization, treatment was discontinued. On the twelfth hospital day the hemoglobin was 5 grams per cent. He was therefore given a transfusion of 500 c.c. of blood and discharged.

DISCUSSION

Sickle-cell anemia is a chronic hereditary disorder generally seen in young persons and largely limited to Negroes or those with some negroid ancestry.[10] It is characterized by a state of mild chronic illness interrupted by episodes of more severe symptoms, the so-called periods of crisis. The cause of crisis is poorly understood. Although the hemolytic element in the disease is well known, it fails to explain all the clinical findings. For example, it is known that crisis occurs in the absence of blood count change or increase in serum bilirubin.[11]

The most striking feature of the disease is the presence of the sickled erythrocytes.[12] Sickling of cells is supposed to initiate capillary stasis which in turn is followed by thrombosis and infarction.[13] However, anatomic studies generally fail to demonstrate thrombosis of a degree sufficient to account for all the clinical findings. According to Kimmelstiel[14] vascular spasm must be considered an important feature of crisis, and in itself may lead to ischemic necrosis without actual thrombosis.

This theory must be given serious consideration in view of the clinical reports given above. The relief of pain of vascular origin by Priscoline has been previously demonstrated in many conditions, notably in peripheral arterial disease and in poliomyelitis. The similar relief of pain or of other obscure and bizarre symptoms in sicklemic crisis bears a close resemblance to the relief obtained in other diseases. The conclusion appears most probable that the mechanism involved is the release of vasospastic phenomenon by Priscoline.

What initiates the intravascular sickling in the first place is difficult to determine. Increased carbon dioxide tension in the blood is known to induce sickling of cells.[15] Perhaps minor infections or toxemias may also induce sickling and hence crisis. It is interesting to note that the crisis in all our patients seemed to have been initiated by such minor infections.

The crisis of sicklemia in the seven patients treated with Priscoline simulated diverse better known conditions. In some instances the diagnosis of sickle-cell disease was known on hospital admission. In other cases the diagnosis had to be made during the hospital stay. The relief of pain or other clinical phenomena through the use of Priscoline was occasionally helpful in determining that the sicklemic crisis was the cause of the syndrome rather than the simulated disease.

According to Watson[16] who has had occasion to observe the effect of Priscoline in a few adults suffering sicklemic crisis, the results were not as spectacular as those seen in the seven children we observed. Perhaps the reason for this is that anatomic changes in adults have progressed to the point where angiospastic relief is no longer obtainable. In our own experience we have noted that Priscoline is somewhat less effective in relieving bone and joint pain than visceral pain. This may be due to the face that generally the viscera have better collateral circulation than do the bones and joints and are more easily affected by vasodilator drugs such as Priscoline. Although sickle-cell crisis is known to be variable and is generally self-limited, we feel

that Priscoline definitely shortened the period of disability in the patients treated.

CONCLUSIONS

1. Seven cases of sicklemic crisis have been presented in which five other disease entities were simulated.

2. Priscoline was administered to these patients, and it proved a useful tool for differential diagnosis and therapy.

3. The theory of vasospasm as a cause of both clinical and anatomic phenomena in sickle-cell crisis was reviewed and extended.

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SOME PROBLEMS IN THE DIAGNOSIS AND TREATMENT OF PULMONARY TUBERCULOSIS IN CHILDHOOD

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IN THE past three years several patients who were admitted to the Children's Hospital of Mont Alto State Sanatorium with a diagnosis of pulmonary tuberculosis presented interesting diagnostic and therapeutic problems. Documentation of these cases appears worth while for the interest of the pediatricians and the physicians who deal with tuberculosis in children.

BRONCHIAL INVOLVEMENT IN PRIMARY TUBERCULOSIS

Through the increased use of bronchoscopy, clinicians have become more aware of the significance of bronchial involvement by tuberculous mediastinal lymph nodes. With the advent of antibiotics and the improvement in the technique of bronchoscopy, it is essential that clinicians recognize this important complication of primary tuberculosis and institute therapy promptly so as to obviate permanent bronchopulmonary damages.

CASE 1.—B. A., a 14-year-old Negro girl, was admitted July 31, 1951. Four months prior to admission, she was hospitalized because of high fever, cough, and night sweats. X-rays revealed greatly enlarged mediastinal nodes on the right side and atelectasis of the right lower lobe. Bronchoscopy showed markedly inflamed bronchial mucosa and complete compression stenosis of the right stem bronchus. An enlarged cervical node was removed from the right side, which proved to be tuberculous on histological examination. She was treated with streptomycin by intramuscular route. PAS could not be given because of intolerance to the drug.

On admission to Mont Alto Sanatorium, no significant change was noticed on the roentgenogram (Fig. 1) as compared with one taken four months previously. Bronchoscopy revealed extensive endobronchial disease. On the medial wall of the right main bronchus there were two whitish, protruding masses measuring approximately 10 mm. in diameter which probably represented protruding lymph nodes. The mucosa beneath these masses was elevated for a distance of 2 cm. On the wall of the intermediate bronchus there was granulation tissue which extended into the middle lobe and lower lobe bronchi, causing partial obstruction of the latter. The granulation was removed piecemeal through the bronchoscope. Histological examination showed caseous tuberculosis. A bronchogram demonstrated marked narrowing of the intermediate bronchus, and Lipiodol failed to penetrate to the middle and lower lobes (Fig. 2).

The patient was treated with streptomycin by aerosol route, in addition to streptomycin by intramuscular route. Three weeks later, roentgenogram showed re-expansion of the atelectatic right lower lobe.

Bronchoscopy three months later showed that the protruding masses in the right main bronchus, noted during the previous examination, were replaced by an irregular friable cauliflower-like mass which bled when traumatized. The intermediate bronchus was narrowed markedly by compression stenosis. The middle and lower

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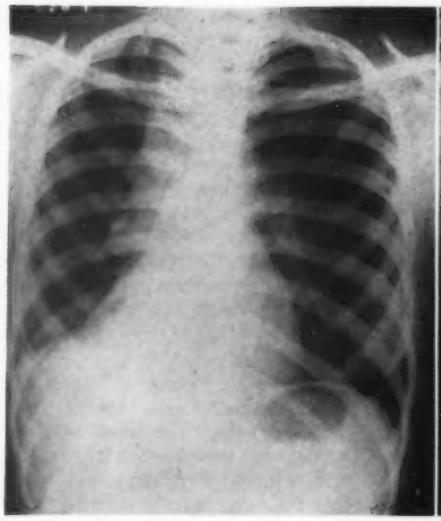

Fig. 1.

Fig. 2.

Fig. 1 (Case 1).—Roentgenogram on admission showing enlarged mediastinal lymph nodes and atelectasis of the right lower lobe.

Fig. 2 (Case 1).—Bronchogram on admission showing marked narrowing of the right intermediate bronchus and nonfilling of the middle and lower lobe bronchi.

Fig. 3.

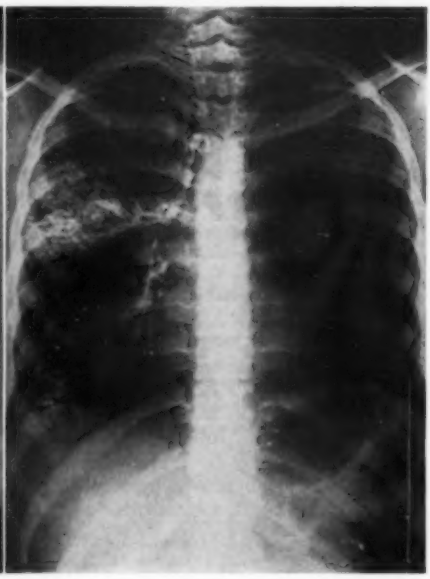

Fig. 4.

Fig. 3 (Case 1).—Roentgenogram taken a year after admission showing diminution in size of the mediastinal lymph nodes.

Fig. 4 (Case 1).—Bronchogram taken fifteen months after admission showing normal right tracheobronchial tree.

lobe bronchi could not be visualized. The patient was maintained on streptomycin therapy. Cough and expectoration soon subsided. There was a rapid gain in weight. Roentgenogram showed gradual retrogression of the mediastinal nodes (Fig. 3).

A year later, bronchoscopy was repeated. The bronchial mucosa appeared normal except for the presence of a small area of granulation tissue on the wall of the intermediate bronchus. This was removed and the site cauterized. Bronchogram revealed a normal tracheobronchial tree (Fig. 4).

child was pale and somewhat cyanotic. The right hemithorax showed very little respiratory motion, and breath sounds were markedly diminished.

Two weeks after admission, cough and dyspnea became more pronounced. X-ray demonstrated extension of the area of cloudiness in the right lung (Fig. 5). Bronchoscopic examination disclosed a granulomatous growth in the right main bronchus and its main subdivisions, causing almost complete obstruction. The growth was removed piecemeal through the bronchoscope. Histological examination proved that

Fig. 5.

Fig. 6.

Fig. 5 (Case 2).—Roentgenogram showing markedly enlarged mediastinal lymph nodes and extensive parenchymal involvement due to atelectasis and/or pulmonary infiltration.

Fig. 6 (Case 2).—Roentgenogram taken three months after admission showing marked reduction in size of the mediastinal lymph nodes and retrogression of the parenchymal changes.

There was no sign of stenosis or bronchiectasis. The patient received a total of 90 Gm. of streptomycin by intramuscular route and 54.4 Gm. by aerosol. She was discharged in good condition in May, 1953.

CASE 2.—G. B., a white boy 3 years old, was admitted on July 17, 1951, with the history of fever and cough of two months' duration. Tuberculin reaction was positive to 0.00002 mg. PPD. Roentgenograms showed enlarged hilar nodes on the right and a zone of cloudiness in the middle third of the right lung field. The right lower lung field appeared emphysematous. The left lung was clear. The

it was a tuberculous granuloma. Culture of the bronchial secretions was positive for tubercle bacilli.

Streptomycin was administered by the aerosol route in addition to the intramuscular administration. Cough and dyspnea soon subsided. Three months later, x-ray demonstrated clearing of the density in the right lung field, and there was marked diminution in size of the right hilar nodes (Fig. 6). However, the breath sounds remained suppressed in the right chest and cough and wheezing were again noticed.

Bronchoscopy was repeated. It revealed recurrence of the granulomatous

growth on the wall of the right main bronchus. After removal of the granuloma, the breath sounds became well transmitted and the wheezing ceased. The patient made an uneventful recovery. He received a total of 58 Gm. of streptomycin by intramuscular route and 20 Gm. by aerosol. The patient was followed up for nineteen months. He grew and developed normally. There was no recurrence of respiratory symptoms.

Comment.—In these two patients the different stages of bronchial involvement by tuberculous hilar nodes were witnessed. These are described by Schwartz[1] and Daly and associates[2] as pressure stenosis, penetration of the bronchial wall, and the perforation of the bronchial wall with subsequent endomural granuloma formation. Removal of the latter through a bronchoscope brought about prompt re-expansion of the atelectatic lobe. In the follow-up bronchoscopy, it is interesting to note the development of fresh granuloma at the site of the penetrating nodes noted at the previous examination, and the appearance of newly developed penetrating nodes on the bronchial wall. These findings not only reveal the pathogenesis of bronchial involvement in tuberculosis, but also explain the recurrence of bronchial obstruction following initial improvement. The importance of repeated bronchoscopic treatment in such cases is stressed by Laff and coworkers[3] and Daly and associates.[2] Prompt removal of the obstructing caseous material and granuloma is believed to lessen the danger of aspiration tuberculous pneumonia and to prevent irreversible bronchopulmonary changes. In both cases streptomycin by aerosol route was given in addition to streptomycin by intramuscular route. An alkaline detergent solution as advocated by Miller and collaborators[4] was used for the aerosol. The response of the endobronchial disease to such combined therapy was satisfactory.

NONTUBERCULOUS CHRONIC PULMONARY INFLAMMATION VERSUS PULMONARY TUBERCULOSIS

CASE 3.—M. M., a 15-year-old white boy, was admitted Sept. 2, 1952, as a case of moderately advanced pulmonary tuberculosis. On admission to the Sanatorium, roentgenogram showed patchy and linear densities scattered throughout both lungs. A bronchogram demonstrated slight distortion of the bronchial tree, but there was no evidence of bronchiectasis. The bronchial mucosa was of normal appearance. All sputum cultures were negative for tubercle bacilli. Tuberculin reaction was negative to 0.005 mg. of PPD on three different occasions. A chest roentgenogram taken three years previously was obtained. No significant change was noticed when compared with the admission film. Sputum cultures and skin tests were both negative for histoplasmosis and coccidioidomycosis. The patient had very little cough and expectoration. Despite the extensive changes on the roentgenogram, the child showed remarkably good pulmonary function as indicated by exercise tests and the ability of holding breath for thirty seconds.

The past history was obtained from the hospitals and clinics where the child had been for treatment. It was revealed that the patient had pertussis at the age of 5, which was followed by repeated bouts of bronchopneumonia over a period of several years. Because of the extensive changes in both lungs which failed to resolve, tuberculosis was suspected. However, the tuberculin test was negative, and no tubercle bacilli could be cultured from the gastric content. Seven years later as a routine procedure prior to

tonsillectomy, an x-ray of the chest was taken. Extensive patchy and linear densities were found bilaterally. Tuberculosis was suspected, but the tuberculin reaction and the sputum test were again negative.

Comment.—The importance of the past history in the diagnosis in this instance is obvious. This is apparently a case of pulmonary fibrosis due to chronic interstitial pneumonia following pertussis. The persistence of the extensive changes on the roentgenogram of the chest has raised the question of pulmonary tuberculosis on several occasions. Our investigations led to the conclusion that the diagnosis of pulmonary tuberculosis could not be substantiated in this case in view of the consistent negative tuberculin reaction and negative sputum tests over a period of ten years. The patient was discharged from the Sanatorium two months after admission on completion of the clinical studies.

CASE 4.—M. P., a 16-year-old white girl, was admitted to the Sanatorium in November, 1951, for pulmonary tuberculosis with the history of chronic cough, loss of weight, and a positive tuberculin test. Examination revealed an adolescent girl, pale and thin, coughing and sneezing frequently. Clubbing of the fingers was conspicuous. X-ray showed diffuse mottling and streaking in both lungs.

After several days of observation, it was noticed that the girl had an enormous appetite. Her bowels moved five to eight times daily. The stools were loose and large, having a very offensive odor. The past history disclosed the fact that the patient has been suffering from chronic cough and recurrent bouts of pneumonia since the age of 6 months. Obstinate diarrhea with frequent large stools dated back to early infancy. Various medicaments were tried, but none seemed to help the diarrhea which was attributed to tuberculosis of the intestine. The child ate much more than her siblings but remained thin. One brother died of intractable diarrhea and pneumonia at the age of 4 months.

Bronchoscopy showed the bronchial secretion was thick and tenacious, culture of which grew staphylococci. The diagnosis of fibrocystic disease of pancreas was entertained. The patient was put on the specific carbohydrate diet as advocated by Haas and Haas.[5] Pancreatin granules and vitamins were given with each meal. The bowel symptoms promptly subsided, and she had a weight gain of ten pounds within three months. As the diarrhea ceased, the cough and expectoration were cut down a great deal. Antibiotic sensitivity test indicated that the staphylococci isolated from this patient was sensitive to Chloromycetin. Chloromycetin was given in the latter part of the hospital course. There was further improvement in the cough. Serial x-ray examinations showed some clearing of the peribronchial infiltration. All sputum tests were consistently negative for tubercle bacilli. The patient was discharged from the Preventorium (Children's Division of the Sanatorium for noncontagious cases) in August, 1952. Examination of pancreatic secretion subsequently made at the Hospital of University of Pennsylvania reported absence of pancreatic enzymes. The diagnosis of fibrocystic disease of pancreas was thus established.

Comment.—This case illustrates the importance of a complete history in the diagnosis of chronic chest disease. The story in this case was so typical of fibrocystic disease of the pancreas that it led to the correct diagnosis, which was subsequently confirmed by laboratory studies and therapeutic response to dietary management and enzyme therapy.

It is remarkable that this girl lived to the age of 16 years without any

enzyme therapy. She was 63½ inches in height and weighed 90 pounds. Secondary sexual characters were well developed.

NONPATHOGENIC ACID-FAST BACILLI FROM THE GASTRIC CONTENT

CASE 5.—J. L., an 11-year-old white girl, was admitted in April, 1950, for pulmonary tuberculosis because of chronic cough and loss of weight of two months' duration. X-ray showed dense infiltration in the right lower lung field. One sputum specimen was positive for acid-fast bacilli.

After admission, the child ran an intermittent fever. The tuberculin reaction was negative to 0.005 mg. of PPD. Bronchoscopy revealed a chronic inflammatory process of the right bronchial tree. Bronchial secretion was purulent and foul. Cultures for tubercle bacilli and fungi were negative. The patient was treated with penicillin and aureomycin. Fever and cough soon subsided. X-ray showed gradual retrogression of the pulmonary infiltration. At this time, one gastric lavage culture reported the growth of acid-fast bacilli. The culture was inoculated into a guinea pig, but it proved to be nonpathogenic. Many cultures of gastric content were done thereafter. No tubercle bacilli was demonstrable. The tuberculin test was repeated four times during the hospital course, all of which were negative. The patient was discharged in good condition. There was no evidence of tuberculosis.

Comment.—The question has repeatedly been raised as to the advisibility of admitting a tuberculin-negative child to a tuberculosis institution. Unless there is a good reason to assume the existence of anergy, a negative tuberculin test is a strong evidence against the diagnosis of active tuberculosis. Finding of acid-fast bacilli in the gastric content of such patient should not cause undue alarm. Considering the frequency with which nonpathogenic acid-fast bacilli can be ingested with food and drink, many tuberculosis institutions disregard the finding of acid-fast bacilli in the gastric content, but depend on the cultural characteristics for the identification of tubercle bacilli. In the case of a negative tuberculin reaction, it is imperative to prove the pathogenicity of such culture in the guinea pigs before it can be taken as a basis for the diagnosis of tuberculosis. If such considerations are taken before sending a child to a tuberculosis hospital, unnecessary exposure of a child to tuberculosis can often be avoided.

NASAL DISCHARGE AS A SOURCE OF ACID-FAST BACILLI IN SPUTUM

CASE 6.—E. S., a 16-year-old white girl, was admitted in January, 1950, for pulmonary tuberculosis, with the history of chronic cough and positive tuberculin reaction.

After admission, x-ray studies showed no sign of active pulmonary disease. However, acid-fast bacilli were found in the sputum on several occasions. Bronchoscopy revealed a normal tracheobronchial tree. Culture from the bronchial secretion was negative for tubercle bacilli.

The patient had been suffering from chronic purulent nasal discharge for many years. The nasal drainage was moderate in amount, having a very foul odor. A smear was made from the nasal discharge and numerous acid-fast bacilli were found. While the acid-fast bacilli were found only intermittently in the sputum specimens, they were consistently present in the nasal secretion and in greater numbers. In the absence of active pulmonary and bronchial disease, there was little room for doubt that the origin of the acid-fast bacilli in the sputum specimens was the nasal secretion.

The acid-fast bacilli in the nasal discharge resemble tubercle bacilli in

morphology and staining characteristics. However, they could not be grown by the method for tubercle bacilli, nor did they produce tuberculosis in the guinea pigs on repeated trials.

Clinically, this was a case of chronic purulent rhinitis. The nasal mucosa was atrophic and pale. There was no ulceration or destruction of the nasal structure which are so commonly seen in leprosy or tuberculosis of the nose. Complete physical examination did not disclose any neurological or dermatological manifestation of leprosy. Serological tests for syphilis were negative.

Comment.—Nonpathogenic acid-fast bacilli are widely distributed in nature which may be ingested or inhaled into the human body. According to Rich,[6] certain acid-fast bacilli have occasionally been discovered from human lesions which appear to be more pyogenic than tuberculogenic. The persistence of a large number of acid-fast bacilli in the nasal discharge in this patient suggested the possibility of this organism being the etiological agent of the purulent rhinitis. However, they were not tubercle bacilli.

That not all acid-fast bacilli found in the sputum are tubercle bacilli, needs no reiteration. This case further reminds us that not all acid-fast bacilli isolated from the sputum are from the lung. One should be on the alert to look for extrapulmonary sources when the existence of active pulmonary tuberculosis is in doubt.

SUMMARY

1. Bronchial involvement by tuberculous hilar nodes is a complication of primary tuberculosis which often leads to bronchiectasis later in life. Such late complication can be prevented by prompt bronchoscopic removal of the obstructing granuloma and the use of antibiotics. Two cases thus treated with success are reported.

2. Chronic nontuberculous infections of the lungs may simulate tuberculosis in symptomatology and roentgenogram appearance. A thorough history is important in the differential diagnosis.

3. Nonpathogenic acid-fast bacilli may be ingested with food and drink; therefore, finding of acid-fast bacilli in the gastric content does not necessarily mean tubercle bacilli.

4. With few exceptions, a persistent negative tuberculin reaction is a strong evidence against active tuberculosis. Mere isolation of acid-fast bacilli from the gastric content of a tuberculin-negative child does not prove tuberculosis without a pathogenicity test of the organism. To avoid the risk of exposing a child to tuberculosis, admission of such cases to a tuberculosis hospital should be discouraged.

5. Not all acid-fast bacilli found in the sputum are from the lung. A case is reported in which the nasal discharge was found to be the source of the acid-fast bacilli in the sputum.

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CONGENITAL PAROXYSMAL TACHYCARDIA

Report of a Case and Review of the Literature

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ALTHOUGH the first case of congenital paroxysmal tachycardia was described over twenty years ago, fewer than a dozen cases have subsequently been reported. On the other hand, noncongenital paroxysmal tachycardias in infancy and childhood have been widely described in the literature and are now frequently encountered by the pediatrician familiar with the clinical picture. However, it is true that some of the cases of paroxysmal tachycardia in early infancy, such as those reported by Nestor and Spece[1] and Hoyer and Lyon,[2] may have represented congenital tachycardias.

The present report was prompted by the observation of a case of congenital auricular flutter in an infant recently born at the University of Minnesota Hospital. It appeared pertinent to report this case, and to review the other cases of congenital arrhythmias other than heart block to see what characteristics they had in common and to compare them with tachycardias occurring after birth. In addition, an attempt has been made to further evaluate the prognosis in these cases by obtaining further information from the authors who reported such cases in the past two decades.

CASE REPORT

M. M., a female infant, was born on March 18, 1952, of a 28-year-old white female whose one previous pregnancy had resulted in the birth of a full-term normal male infant. Pregnancy had been relatively uncomplicated, although the mother had taken Banthine (a parasympathetic depressant) in low dosage for about ten days two months prior to term because of gastrointestinal difficulties. Approximately three weeks before the birth of M. M., the attending obstetrician had noted an irregular fetal heart rate, and this was again noted shortly before birth. Labor occurred spontaneously, and the membranes were artificially ruptured. Delivery was uncomplicated, and the total duration of labor was five hours.

Examination at birth revealed a slightly dyspneic 7¼ pound female infant whose extremities were minimally cyanotic. The heart appeared greatly enlarged and there was a prominent cardiac thrust. No heart murmurs could be heard. The heart rate was irregular and varied in rate from 50 to 180. An electrocardiogram taken two hours after birth showed the presence of auricular flutter with a variable block, the ventricular rate being about 190 (Fig. 1). A roentgenogram taken at this time showed very marked enlargement of the heart of a rather diffuse type.

The infant was given oxygen, and despite the marked cardiac enlargement and cardiac irregularity, appeared to be in fairly good condition. Digitoxin therapy was started when the infant was 19 hours old. A digitalizing dose of 0.18 mg. of digitoxin was given intramuscularly in divided doses over a period of eight hours. This was followed by a daily maintenance dose of 0.02 mg. There was no significant change in the cardiac rhythm until the fifth day of life, when the irregularity was replaced by a regular rate of 100 to 116. During this time the infant

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took feedings well, and was gaining weight at the time of the rhythm conversion. On the seventh day, an electrocardiogram showed a normal rhythm, broad flat-topped P waves in the first standard lead, and digitalis effects. Roentgenograms on the same day showed moderate cardiac enlargement, but on the following day showed a normal-sized heart. Oxygen therapy was discontinued when the change to normal rhythm occurred. The infant was discharged from the hospital on the eighth day of life. When seen again three days later, the infant appeared normal, although the electrocardiogram still showed abnormal P waves. Digitoxin was then discontinued. One week later, examination revealed a soft systolic murmur over the base of the heart, and occasional premature beats were heard. Later examinations at the age of 1 month and 10 months showed normal physical, electrocardiographic, and roentgenographic findings.

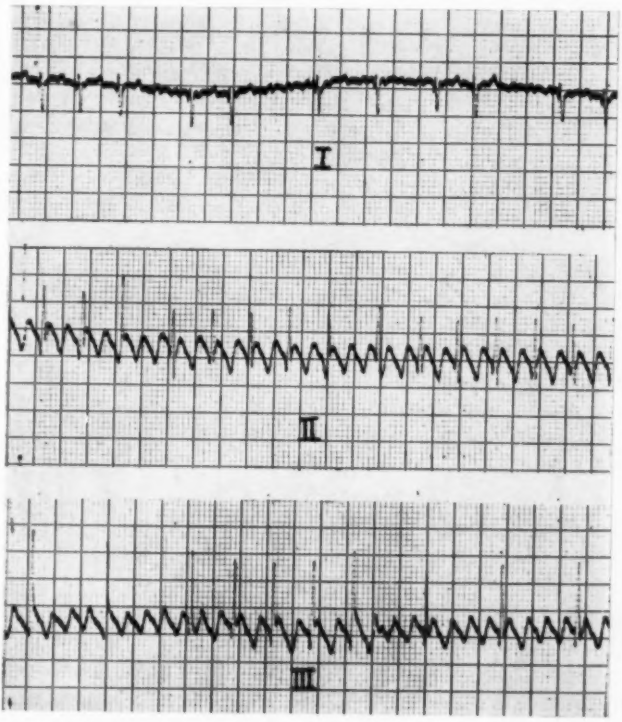

Fig. 1.—Electrocardiogram taken two hours after birth. Standard leads.

REVIEW AND DISCUSSION OF LITERATURE

As can be seen from Table I, tachycardias have been detected as early as the sixth month of pregnancy, although usually the observation has been first made in the last month. The fetal heart rate has usually been described as rapid, irregular, or rapid and irregular. In some of the pregnancies various complicating factors were present, but there would appear to be no common factor that might be considered to have etiological significance.

TABLE I

AUTHOR	TIME OF DETECTION	SEX AND WEIGHT	TYPE OF ARRHYTHMIA VENTRICULAR RATES	RELATED FINDINGS	SPECIFIC THERAPY	DURATION OF ARRHYTHMIA	FOLLOW-UP
Carr and McClure[4] 1931	3 wk. before birth. Irregular rate of 160	F 10-13½	ECG 2nd da. Rate of 192 with flutter and irregular block	Enlarged heart on 1st da. Slight cyanosis on crying	None	10 da.	Normal at 4 mo.
Tollas[5] 1933	24 hr. before birth. Rapid irregular rate of 200	M ---	No ECG	----	None	4 wk.	Normal at 6 wk.
Sherman and Schless[6] 1934	8th month. Rapid rate of 190	M 7½	ECG 2 mo. Rate of 232 with flutter and 2:1 block	Enlarged heart on 2nd da. Dyspnea and cyanosis at 1st and 4th mo.	Digitalis at 4-6 wk. and again at 4 mo.	4 mo.	Normal at 5 mo.
Tarnower and Lattin[7] 1942	8th month. Irregular rate of 190	F 8-12	ECG 1st da. Rate of 195 with flutter and irregular block	Color poor	Carotid pressure unsuccessful. Then digitalis	3 da.	*Normal at 10 yr. No recurrences
Garvin and Kline[8] 1947	6th month. Rapid rate of 200-250	F 8-1½	ECG 2nd da. Supraventricular tachycardia of 200	Enlarged heart on 2nd da.	Digitalis for 3 da. Then quinidine	Intermittent for 3½ mo.	*Normal at 5½ yr. No recurrences
Taussig[3] 1947	"Rapid rate prior to birth"	F ---	Flutter with block	----	Digitalis at 7th mo.	7-12 mo.	Evidence of interatrial septal defect at 5 yr.
Stadler[9] 1948	8th month. Rapid irregular rate of 136-208	M 7-13	ECG 7th da. Rate of 135 with flutter and 2:1 block	Enlarged heart on murmur 1st da. Sclerema 5th da.	Vagal pressure not successful. Then quinidine. Digitalis started 22nd da.	3½ wk.	*Normal at 3 yr. No recurrences
McLean[10] 1952	Several weeks before birth. Rapid and irregular rate	M 8-14	ECG 3rd da. Rate of 200 with flutter and irregular block	Heart enlarged at 1 mo.	Digitalis. Then quinidine. Then digitalis	1 mo.	Normal at 1 yr.
Sancetta, Redding, and Haubrich[11] 1952	Several hours before birth. Tachycardia of 240	M 7-11	ECG at 1 hr. Supraventricular tachycardia of 222	----	Bilateral carotid pressure successful	1 hr.	Normal at 9 mo.
Anderson and Adams	3 wk. before birth. Irregular rhythm	F 7-3	ECG at 2 hr. Rate of 190 with flutter and irregular block	Slight dyspnea and cyanosis, enlarged heart, 1st da.	Digitalis	4 da.	Normal at 1 yr.

*Personal communication.

The high average birth weight of 8 pounds 6 ounces, with no infant weighing less than 7 pounds 3 ounces, is rather striking, however. Despite cardiac enlargement in the majority of cases, none of the infants was considered to be in critical condition at any time and none died. This was true in spite of a persistence of the abnormal rhythm for as long as seven months, the average duration being a month or more. Little can be summarized regarding treatment, except to say that the more striking results were those following the use of some type of digitalis. In two cases return to normal rhythm occurred spontaneously, and in several others the return to normal rhythm may have been unrelated to therapy. The prognosis was uniformly good. Except for the case reported by Taussig,[3] which was thought to involve an auricular septal defect, the infants showed no evidence of cardiac defects. Perhaps the preponderance of cases of auricular flutter accounted greatly for the excellent immediate prognosis in these cases, for as a number of authors have suggested, the occurrence of flutter is probably a less serious situation than other types of paroxysmal tachycardia in infants.

The most suitable series of noncongenital paroxysmal tachycardia cases for comparative purposes is that of Nadas and associates,[12] particularly their group of twenty-five cases in infants under the age of 5 months. Table II draws attention to those features which vary most in the two groups, and which might suggest some basic differences.

It is interesting to note that Nadas's one female case developed her arrhythmia on the first day of life. Although the differences between the two groups with respect to sex incidence and type of arrhythmia appear statistically significant, their real meaning remains unexplained, and conjectures at this time would not appear justified. Suffice it to say that there is suggestive evidence that congenital paroxysmal tachycardia may be etiologically different from later developing tachycardias. So far as treatment is concerned, the preferred drug appears to be digitalis in the dosage recommended by Nadas and his group. They recommend for infants a dosage of digitoxin or its equivalent of 0.06 mg. per 1.0 kilogram of body weight; maintenance dose one-tenth of this for a period of a week or more. In cases where the obstetrician is able to diagnose a fetal paroxysmal tachycardia there would seem to be no good reason for altering the usual management of labor and delivery.

TABLE II

	CONGENITAL CASES (FROM TABLE I)	INFANTS UNDER 5 MONTHS (DATA OF NADAS, ET AL.)
Number	10	25
Sex: Females	5	1
Males	5	24
Arrhythmia:		
Auricular flutter	8	7
Supraventricular tachycardia	2	17
Ventricular tachycardia	0	1

SUMMARY

A case of congenital auricular flutter is described, and a review is made of the nine previously described cases of congenital paroxysmal tachycardias. The prognosis is uniformly good, with all cases showing a reversion to normal rhythm. Only one case was associated with a known heart defect. Recommended drug treatment is digitoxin in a dosage of 0.06 mg. per 1.0 kilogram of body weight. Congenital paroxysmal tachycardia differs from that commencing in the early months of infancy by its equal occurrence in males and females and by the preponderance of flutter arrhythmias.

The authors appreciated the kindness of Drs. Edward M. Kline, Harold E. Stadler, and Herman Tarnower in furnishing additional information about their patients.

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ADDENDUM

Since this article was submitted for publication, an additional case report has been located in the literature, that by G. T. Hedberg.* He reported congenital auricular flutter in an 8 pound 13 ounce male infant. The arrhythmia was first noted twenty-six days before birth, and persisted for two months after birth. Digitalis therapy was apparently responsible for the conversion to normal rhythm. The infant appeared normal at the age of 8 months, although the electrocardiogram at that time showed some evidence of intraventricular conduction block.

*Hedberg, G. T.: Foetal Arryhythmia—Congenital Auricular Flutter, Acta obst. et gynec. scandinav. **25**: 392, 1945.

THE USE OF HIBICON IN THE TREATMENT OF EPILEPSY

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IN 1952, Hawkes[1] and Kaplan and Maslanka[2] independently reported good results with the use of Hibicon (N-benzyl-β-chloropropionamide) in the treatment of epilepsy. Some of their patients were treated with Hibicon alone, others were given Hibicon in addition to a barbiturate or diphenylhydantoin sodium (Dilantin). These investigators observed beneficial results primarily in major motor epilepsy. A few of their patients with psychomotor epilepsy were also favorably affected. No significant untoward reactions were encountered in any of these patients. The purpose of this paper is to report our findings in forty-nine epileptic patients who were treated with Hibicon.

MATERIALS AND METHODS

Hibicon was administered to twenty-eight patients who had previously been observed in the epilepsy clinic of the Johns Hopkins Hospital for at least three years. Each of these patients had been treated with maximum dosages of phenobarbital, Dilantin, or Mesantoin, both singly or in combination, without benefit. Hibicon was administered to each of these patients in addition to the same dosage of the previous ineffective medication. Six additional patients, who had never received any form of anticonvulsant therapy, were given Hibicon alone. Fifteen other patients, who had previously been treated by other physicians with the recommended dosage of Hibicon without benefit, were referred to our epilepsy clinic for further study and treatment. We prescribed phenobarbital for seven of these patients, Dilantin for four, and Mysoline for the remaining four.

All of these forty-nine patients suffered with frequent (daily or weekly) major motor seizures. Thirty of them had cryptogenic (idiopathic) epilepsy, nineteen had secondary (organic) epilepsy. Thirty-one of these patients ranged in ages from 7 to 14 years, eighteen were over 15 years of age.

The dosage of Hibicon administered to our patients was as follows: for children, 250 to 500 mg. three to four times a day, and for adults, 1 to 2 Gm. three to four times a day. This is the same dosage of Hibicon advocated by Hawkes.[1] Hibicon therapy was continued in each of our patients for at least three months.

RESULTS AND DISCUSSION

In a few of our patients there was a temporary decrease in the frequency of seizures but in no case did the administration of Hibicon completely control the seizures. Our findings differ from those of Hawkes[1] and Kaplan and Maslanka.[2] Hawkes[1] reported that "adequate control of convulsive seizures was achieved in 39 of 59 patients given clinical trial with Hibicon, supplemented by a barbiturate in 11." Kaplan and Maslanka[2] stated that "great improvement" or "slight improvement" was obtained in 70 per

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cent of thirty-one patients treated with Hibicon. These investigators, however, did not mention the actual number of their patients in whom seizures were completely controlled. Our results do not support Hawkes'[1] statement that "Hibicon may be employed in the treatment of chronic epilepsy with an efficacy which is comparable to that of other modern anticonvulsant drugs." In sixteen of the forty-nine patients included in this report, seizures were subsequently completely controlled with the use of phenobarbital in eight, Dilantin in five, and Mysoline in three.

It is also of definite importance that Hibicon is a very costly drug. Its cost is at least twenty times that of phenobarbital and approximately five times that of Dilantin. This is a very important factor in the treatment of epilepsy, since most patients must take daily medication for many years. Another disadvantage of Hibicon is that it is marketed in a very large, completely sealed, capsule which is extremely difficult to administer to young children.

In conclusion, our findings indicate that Hibicon is of very little, if any, value in the treatment of major motor epilepsy. A similar opinion has also been expressed by Arieff.[3]

SUMMARY

Hibicon was administered to forty-nine patients who suffered with frequent major motor epileptic seizures. In none of these patients were the seizures completely controlled.

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ACUTE GASTROENTERITIS

A Review of 518 Cases Treated at the Hospital for Sick Children During 1951 and 1952

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AFFECTIONS of the gastrointestinal tract of infants and young children have attracted the attention of medical practitioners since the dawn of recorded medicine. The well-known association of teething and digestive upsets was recorded by physicians of the Hippocratic school. The syndrome known at present as acute gastroenteritis or acute infantile diarrhea was recognized as a leading cause of death both in Europe and on this continent as early as the eighteenth century. On this side of the Atlantic, the disease was first recognized by Benjamin Rush who described a severe, often fatal, malady affecting young children during the summer months, in the cities and towns of the Atlantic coast. This he called cholera infantum because of its resemblance to Asiatic cholera then ravaging the cities of Europe.

Various theories, some logical and many not, were advanced to explain the nature of this disease. Heroic and sometimes harmful measures were instituted to effect cures, but the death rate remained alarmingly high. Recognition of the harmful effects of water and electrolyte losses in vomiting and diarrhea, and the understanding of practical replacement therapy, has reduced the mortality of gastroenteritis considerably in the past thirty years. However, it still remains a baffling disease, accounting for considerable severe illness among our infant population.

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MATERIAL STUDIED

This present series consists of 518 consecutive cases admitted to this hospital in the eighteen-month period from July, 1951, through December, 1952. There were considerably more than this number of cases admitted to hospital with this diagnosis but it was found that "gastroenteritis" was applied loosely to various types of digestive disturbances. These included improper feeding, colic, hypertonicity, and milk allergy. It was also applied to physiological variations in stool patterns, and to the acute exacerbations of chronic gastrointestinal diseases such as celiac syndrome or fibrocystic disease.

Infants who were admitted to hospital for some other illness, but who contracted gastroenteritis while in hospital, also were included in this series. The very large number of infants treated through our outpatients service were not included in this study unless they were admitted to hospital.

SOCIOECONOMIC FACTORS

Authors have consistently stressed the importance of poor environment in relation to gastroenteritis. Twenty years ago, Johnston and associates[1] reported that of 172 patients admitted to this hospital with this illness, only

two were treated on the private wards. While one is often struck with the obviously poor care some of our patients have received prior to admission, the victims of this malady are not wholly confined to the underprivileged infants in our population. Fully 42 per cent of the infants studied were admitted to the private services of this hospital and presumably came from homes of adequate financial status. Most homes of infants admitted were visited by Public Health Nurses and their reports varied from good to poor housing and maternal care.

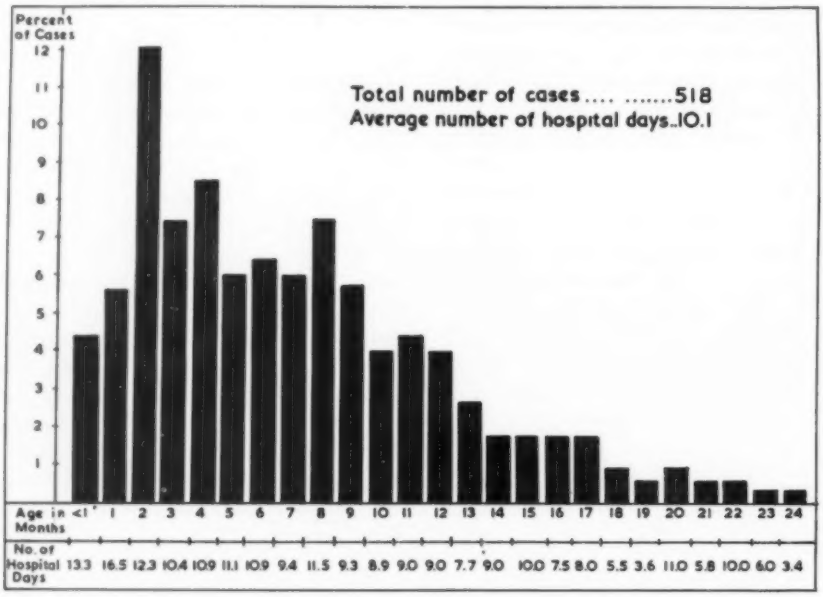

Fig. 1.—The age incidence and relationship of age to length of hospital stay.

AGE INCIDENCE

Gastroenteritis is a disease of early infancy. In this series, the youngest patient was 4 days and the oldest 24 months. The great preponderance in the first four months of life is evident in Fig. 1. In this age group we found a severer form of the disease with a higher mortality rate. Over 75 per cent of our fatal cases were in this age group. The peak incidence of our series was at 2 months, 50 per cent of the cases were under 6 months, and the disease was rarely found after the seventeenth month of life.

SEASONAL INCIDENCE

The name "summer diarrhea" by which this disease was called for many years attests to its seasonal incidence. In former years September was a month to be dreaded in Toronto because both the morbidity and mortality of the disease soared,[2] and this hospital was filled to capacity with infants ill with gastroenteritis. Most investigators have found a definite seasonal variation, many in late summer

and early fall[3] and some in spring.[4] The seasonal variation once so well defined at this hospital is now not as apparent. The illness is seen throughout the year, and in this series, the cases occurring in summer only slightly exceeded those during the winter months.

BREAST FEEDING

It is generally accepted that breast-fed babies are less likely to contract this disease than those infants fed artificial formulas. Of the first 300 infants studied in this series, only eleven or 3.7 per cent had been breast fed for longer than two months, and only two were being breast fed at the onset of their illness.

ETIOLOGY

The lack of unanimity of opinion as to the etiological factors responsible in this disease is proof in itself of the challenge gastroenteritis offers. Discussion at present revolves mainly around the relative importance of enteral and parenteral infection as the causative factor in the etiology of this disease.

TABLE I. THE ETIOLOGY OF CASES

	NO. CASES	PER CENT
1. Parenteral infection	207	40.0
Upper respiratory infections	123	59.0
Otitis media	46	22.0
Pulmonary infections	14	7.0
Pyogenic skin diseases	13	6.5
Other parenteral infections	11	5.5
2. Known enteral infection	23	4.4
Salmonella and Shigella	11	48.0
Thrush	12	52.0
3. Suspected enteral infections	288	55.0

In our present series 40 per cent of the patients were admitted with evidence of parenteral infection. These were mainly upper respiratory infections, acute otitis media, pulmonary infections, and pyogenic skin diseases. This sizable relationship of gastrointestinal symptoms to parenteral infection is in keeping with that found in other series.[5, 6, 7, 8] The relationship of these parenteral infections to the gastroenteritis is most indefinite. Diarrhea and respiratory symptons may be the result of a single infection resembling the "flu" of adults.

The theory, first promulgated in Europe by Renaud and Preysing, that hidden mastoid infections were a cause of gastroenteritis, was widely accepted on this continent following the work of Marriott. The validity of this theory was questioned at this hospital by Brown and Wishart[1] and in other centers, after a multitude of myringotomies and mastoidectomies had been done with no appreciable change in the course of the gastroenteritis. In this series mastoidectomy was performed on one patient in quest of a hidden focus of infection, with negative results. Intercurrent infections, so prevalent and devastating in earlier investigations, were insignificant in our cases; probably this was due to antibiotic therapy.

In only 4.4 per cent of our patients was a proved enteric pathogen recovered from the stools. Except for Levinson and Raycraft's[3] series wherein 31.5 per cent of their cases were associated with Salmonella or Shigella infection, most investigations have reported these organisms in only a small number of cases.[10, 11] In the remaining 55.6 per cent of our infants no known etiological agent could be identified. These were termed suspect enteral infections.

The absence of a definite pathogenic microorganism in such a large proportion of our cases is a finding shared by all recent series. This has given impetus to numerous investigations. A possible viral agent responsible for gastroenteritis has been isolated by several investigations. The disease was transferred to mice by Lembke and co-workers,[12] to calves by Light and Hodes[13] and Buddingh and Dodd,[14] and to human volunteers by Reiman and associates[15] and Gordon and collaborators.[16] Various bacteria, also found in the stools of normal unaffected individuals, have been found in localized outbreaks of gastroenteritis. In the past, the *Streptococcus fecalis*, the Paracolon and Proteus groups of organisms, and *Pseudomonas aeruginosa* were thought to have been causative organisms.

Pathogenic varieties of *Escherichia coli* variously known as type 055,-0111B$_4$, and *E. coli neapolitanum* have been pursued in recent epidemics. Following the original work of Adam, and of Cziglany,[17] these varieties of the colon bacillus have been reported from the United States,[18, 19] Sweden,[20] Great Britain, Denmark, Holland, and Israel.[4, 21]

It is possible that invasion of the gastrointestinal tract by any new organism, pathogenic or not, may result in diarrhea before the host becomes acclimatized.

Stool cultures were obtained from 66.3 per cent of the cases in this series. Only 3.1 per cent of the organisms cultured were generally accepted pathogens. Studies for isolation of viruses or identification of the serotypes of *E. coli* were not undertaken.

The staphylococcus has been implicated in several outbreaks of diarrhea. Resistant strains of staphylococci have developed in patients treated with Aureomycin and Terramycin and these have been held responsible for severe gastrointestinal upsets.[22] In our series, staphylococci have been isolated from 100 or 28.2 per cent of the stools cultured. However, when these cases were compared with those in which other common enteric organisms were cultured, no greater proportion of severe or lengthy illness

TABLE II. THE BACTERIOLOGY OF THE STOOLS

	NUMBER	PER CENT
Stool cultures taken	355	66.3
No report available	35	10.0
"Usual fecal flora" reported	72	20.0
Proved pathogens		
Salmonella typhimurium	3	3.1
Salmonella para-typhi B	2	
Salmonella oranienburg	1	
Unspecified Salmonella	1	
Shigella sonnei	3	
Shigella alkalescens	1	
Suspected pathogens		
Staphylococcus pyogenes	100	28.2
Proteus morganii	75	21.1
Proteus mirabilis	57	16.0
Pseudomonas aeruginosa	36	10.2
Paracolobactrum coliforme	17	4.5
Paracolobactrum aerogenoides	16	4.5
Candida albicans	4	
Proteus vulgaris	3	
Streptococcus entericus	2	
Streptococcus viridans	1	
Paracolobactrum intermedium	2	
Paracolobactrum bethesda	1	
Anaerobic paracolon	1	

TABLE III. RELATIONSHIP OF FLORA TO SEVERITY

	DURATION OF HOSPITAL STAY		
	UNDER 10 DAYS (%)	10-15 DAYS (%)	OVER 15 DAYS (%)
Staphylococcus aureus	49	23	28
Proteus morganii	40	24	36
Proteus mirabilis	37	26	37
Pseudomonas aeruginosa	44	33	23

was encountered. Ninety-seven patients in this series received Aureomycin or Terramycin during their illness, but we were unable to find any severe or protracted illness in which staphylococci were cultured from the stools. It is also of interest to note that in none of our cases thought to be due to *Candida albicans* did the patients receive these antibiotics.

CLINICAL PICTURE

For the purpose of study, we have divided our cases into four groups according to their state of hydration. One hundred seven (20.5 per cent) infants were not clinically dehydrated when first seen.

One hundred fifty-three (29.5 per cent) of these infants were found to be mildly dehydrated. They were irritable; the skin, mouth, and tongue were dry, the lips unduly red, and the eyes noticeably bright. Thirst was a prominent symptom, but these children were prone to vomit if they drank fluids of any sort. Another 166 (32.2 per cent) were moderately dehydrated. They were alternately irritable, or drowsy and apathetic. The skin was dry and had lost its usual elasticity. The mouth and tongue were dry and coated. The fontanel was sunken and the eyes bright or glassy. The abdomen had a characteristically doughy feel and the extremities were cool and limp. Tachycardia was frequent and the respirations were usually of an acidotic type.

Severely dehydrated patients accounted for ninety-two cases (17.8 per cent) in this series. These infants were very listless and often apparently comatose, crying feebly only when stimulated. They were shocked with cold, mottled, flaccid, cyanotic extremities. Their eyes were rolled up and sunken. Respirations were either slow and shallow, or if acidosis was severe, deep and gasping. The heart sounds were rapid and distant, but in those infants admitted in extremis, the cardiac rate was more often slow and irregular.

TREATMENT

The treatment of infants suffering from gastroenteritis consisted of five stages: (1) initial resuscitation and treatment of shock, (2) correction of water and mineral loss, and acid-base imbalance, (3) maintenance of water and mineral equilibrium, (4) antibiotic therapy and specific antidiarrheal medication, (5) resumption of milk feedings and convalescence. There are no set rules for the treatment of gastroenteritis at this hospital; each case is handled individually by the house and attending staffs. Because of this there were certain variations in the methods used, which offered an opportunity for comparison.

INITIAL RESUSCITATION AND TREATMENT OF SHOCK

Infants admitted to hospital in a dehydrated state were given intravenous fluids shortly after admission. Where shock was present, whole blood or plasma was administered in a dose of 20 ml. per kilogram of body weight over a period of one to two hours. Scalp veins or cut-downs on the long saphenous vein were used as transfusion sites. Fifty-seven (11 per cent) patients, received either whole blood or plasma shortly after admission.

We have occasionally used adrenal cortex extract intravenously in the treatment of the shocked infant. Eleven (2 per cent) infants received

this extract during the initial stages of therapy. We had no fatalities in this group but the number was too small to judge the value of this material in therapy. Emdin[23] has recently used this extract in conjunction with potassium chloride in the treatment of eighty-three patients with good results.

CORRECTION OF WATER AND MINERAL LOSS

Seventy-eight per cent of all patients with gastroenteritis were given intravenous fluids on admission to hospital. The solution most frequently used was two-thirds 5 per cent glucose and one-third normal saline solution. This provides a hypotonic saline solution with adequate carbohydrate. We have found this to be the solution of choice except in those cases of hypertonic dehydration with hypernatremia and hyperchloremia. In these cases we have used 5 per cent glucose in distilled water as the hydrating solution.

Our goal in dehydrated infants was to replace water loss and to restore hydration within the first twenty-four hours. We have found that one cannot give intravenous fluids by a set formula based on the infant's body weight and expected water loss alone: clinical judgment of the initial state of the infant and the rate of improvement must guide the course of therapy. In general, we give 75 to 150 ml. per hour until the hydration begins to improve and then reduce the rate of intravenous to 30 to 50 ml. per hour.

We have routinely used M/6 sodium R lactate in those patients who had clinical evidence of acidosis and a depression of the carbon dioxide combining power below 25 vol. per cent. In a dose of 1.6 ml. per kilogram of body weight per volume per cent we give sufficient solution to correct the acidosis to 40 vol. per cent, and expect that with restoration of water balance the kidney can complete this correction.

MAINTENANCE OF WATER AND MINERAL EQUILIBRIUM

After hydration has been restored, we have been accustomed to continue intravenous therapy, usually for a period of three to five days. This has depended upon the amount of food taken by mouth and the volume of fluid lost through diarrhea, or until the likelihood of relapse with renewed dehydration has ceased. Again we have found that two-thirds 5 per cent glucose in one-third normal saline has been the most practical solution to use.

Recognizing the importance of intracellular loss of potassium and the dangers of prolonged hypopotassemia, we have given potassium replacement therapy as described by Darrow and co-workers[11, 24, 25] to 40 per cent of our infants. We have used Darrow's solution intravenously or subcutaneously, or have given potassium by mouth as potassium chloride or as Darrow's solution diluted 1:2 with sugar solutions. In dosages of 80 ml. per kilogram per day intravenously, we have been able to restore serum potassium levels in most patients within a few days, but with no greater success than by the oral route, provided that vomiting has subsided.

In 25 per cent of our cases we have also used the subcutaneous route of administration of fluids. This has usually followed intravenous therapy during the more acute phase of the illness. This method of therapy has

been used less frequently since the widespread acceptance of the scalp vein as an easy and innocuous site for fluid replacement. We have also used continous interstitial therapy with hyaluronidase in a few patients, but have found no advantage to this method in the usual patient, and have now confined its use to infants whose veins are inaccessible.

In addition to the use of blood in the initial phase to treat shock, we have used whole blood liberally in the further treatment of our patients. Two hundred fifteen (41.4 per cent) infants received blood transfusions during their hospital stay. Blood was given for the treatment of anemia frequently seen after rehydration had been completed, in those cases where the serum proteins were low, and in other cases for its general rehabilitative effect. Fourteen (2.1 per cent) infants were given plasma during treatment.

ANTIBIOTIC AND DRUG THERAPY

In a disease of obvious infective nature one can expect that numerous clinical trials with many antibiotic and chemotherapeutic agents would be undertaken, despite the fact that no causative agent has yet been generally accepted. All of the common drugs have been tried in the treatment of gastroenteritis and most have undergone extensive investigation. However, for each drug there have been both encouraging and disappointing reports. To date there is no conclusive evidence that this disease has been materially affected by any antibiotic.

In this series we have employed the various antibiotics in combination or alone. Penicillin, the sulfonamides, Aureomycin, Terramycin, or chloramphenicol have been used in 84 per cent of the cases. All cases of gastroenteritis due to Shigella or Salmonella were treated with sulfonamides or chloramphenicol with no relapses. However, our results in the great majority of cases not associated with these organisms were disappointing.

Where the infant was well hydrated, there was a slight reduction in the hospital stay for those patients receiving antibiotics, when compared to those not receiving this type of therapy. This difference was not dramatic and probably represented a diminution of intercurrent infection. In those cases where dehydration was present, i.e., cases of greater severity, antibiotic therapy of any type or combination had no significant effect. Indeed it can be seen in Table IV that those infants receiving multiple drugs had a protracted hospital course. This does not imply that the antibiotic therapy was responsible for the diarrhea, but that the illness continued in spite of several changes of antibiotics. We

TABLE IV. ANTIBIOTIC THERAPY

DRUG	TOTAL CASES	AVERAGE NO. DAYS DIARRHEA	AVERAGE NO. DAYS HOSPITAL	DRUG
A. Normal Hydration				
Penicillin	18	2.4	6.0	3.8
Aureomycin	12	2.5	7.0	4.5
Penicillin and streptomycin	10	7.4	11.5	6.8
No drug therapy	28	3.3	9.6	--
B. Dehydration				
Penicillin	86	4.0	8.9	5.9
Aureomycin	13	5.1	9.2	4.5
Terramycin	25	7.0	11.8	5.8
Penicillin and streptomycin	73	4.4	10.2	6.1
Penicillin and Aureomycin	20	10.2	14.6	9.0
Multiple drug therapy	53	11.4	18.0	12.3
No drug therapy	62	4.1	9.0	--

feel that antibiotic therapy may be useful in the treatment of an underlying parenteral infection, or for the prevention of intercurrent infection but has no direct effect upon the diarrhea.

It has been the practice at this hospital to use camphorated tincture of opium routinely in most cases of gastroenteritis to control the diarrhea. This has been used alone or in combination with chalk or bismuth compounds. To evaluate the effect of this compound we have compared the course of fifty infants who received camphorated tincture of opium with thirty-two others who did not receive this medication. These cases were comparable in that they were of equal severity, and the patients received no other form of therapy, including antibiotics. With adequate dosage we found no real effect of this drug on the duration of the diarrhea or the subsequent hospital course, although these children seemed to be less irritable while receiving the opiate.

TABLE V. THE EFFECT OF OPIATES

	NO. OF CASES	NO. OF DAYS	
		DIARRHEA	HOSPITAL
Medication given	50	5.1	10.0
No medication given	32	3.4	8.0

RESUMPTION OF FEEDINGS

In the past it has been our practice to start small frequent feedings of clear sugar solution almost immediately following admission to hospital. Recently the practice of giving nothing by mouth from upward of twenty-four hours has been advocated to shorten the period of vomiting.[7, 10, 24] We decided to test this hypothesis and gave seventy-four of our infants admitted with vomiting as well as diarrhea nothing by mouth for twenty-four hours. When the length of time these children continued to vomit or have loose stools was compared with a comparable group of 237 infants who were fed frequent small amounts of clear sugar solutions, no significant difference could be found. This initial feeding supplies water, carbohydrates, and minerals, and does not seem to be detrimental to the course of the illness.

The time-honored tradition has been to initiate milk feeding cautiously and in dilute form during convalescence. Recently Chung and Holt[26] have attacked this theory and have treated infants in the United States and in Czechoslovakia initially with full feedings with no untoward effects. We have been unable to confirm these findings. Patients who have been fed full-strength feedings immediately had a higher incidence of relapse than those infants to whom dilute feedings were first given. It is our practice to give nothing but clear sugar solutions until the stools begin to form and then to start dilute milk feedings. We employ, in the majority of infants, either protein milk or skim milk diluted to give a feeding of 30 to 60 calories per kilogram, but have also had success with diluted 2 per cent evaporated milk mixtures of similar caloric value. As the patient progresses, this mixture is increased in strength to provide 80

TABLE VI. THE EFFECTS OF EARLY FEEDINGS

	NO. OF CASES	NO. OF DAYS	
		VOMITING	DIARRHEA
Initial starvation	74	2.5	5.7
Initial feedings of sugar solutions	237	2.8	5.6

All patients admitted with history of vomiting

to 100 calories per kilogram. We have been unable to find any significant differences in our infants treated with these varieties of milk provided that the dilution has been employed.

It has been our practice to add carbohydrate to the milk feeding and to start solid foods in small amounts when the stools become formed. The solid foods are increased slowly, but if a recurrence of diarrhea is encountered, a return to basic milk feeding is made.

repeated in those patients receiving alkali therapy and a rise in these values was uniformly found. We encountered only one patient with alkalosis due to severe potassium depletion; in this infant the alkalosis was corrected by the use of potassium orally and intravenously.

Measurements of serum sodium, potassium, and chloride levels were obtained on ninety-three patients. All were either severely or moderately

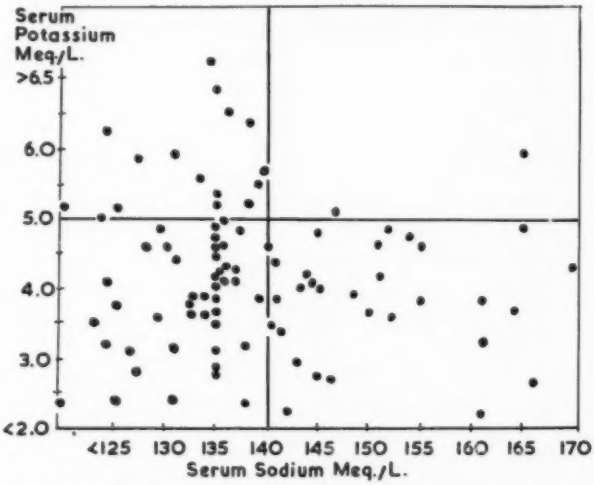

Fig. 2.—The relationship of serum potassium and sodium values.

BLOOD CHEMISTRY DETERMINATIONS

Carbon dioxide combining power and pH measurements were obtained on 100 of our dehydrated patients. We found the degree of acidosis to be closely related to the severity of the dehydration. Moderately dehydrated infants had an average carbon dioxide combining power of 33.4 vol. per cent and an average pH of 7.30, while the severely dehydrated infants had an average carbon dioxide combining power of 24.0 vol. per cent and a pH of 7.17. These determinations were

dehydrated. Many of these measurements were repeated at intervals following rehydration. The usual picture was one of hypotonic dehydration and acidosis. In these cases the serum sodium levels were either low or appeared to be within normal limits due to severe dehydration with contraction of the extracellular space. These values ranged from 120 to 150 meq. per liter. The plasma chloride levels were elevated with values of 100 to 130 meq. per liter. Following intravenous therapy and restoration of

hydration, there was a rise in sodium and a fall in chlorides to more normal levels.

In eight patients, a picture suggesting hypertonic dehydration was present with hypernatremia and hyperchloremia. These were all severely dehydrated and acidotic infants. Sodium levels ranged from 152 to 178 meq. per liter and the chlorides from 115 to 140 meq. per liter. Following hydration with glucose in distilled water solutions, these levels fell to normal.

We were unable to find any relationship between serum potassium levels and the clinical course of the patient, either with regard to the diarrhea, or the length of hospital stay. A few infants admitted with hypopotassemic levels received adequate potassium replacement therapy, followed by a rise in serum potassium levels, but went on to have prolonged diarrhea for no known reason. Others, in contradistinction, were admitted with normal or already deficient values, received no potassium therapy, had a fall in serum concentrations, even to dangerously low levels, but recovered in the average time. We did, however, endeavor to restore or maintain potassium levels in most infants and used potassium replacement therapy in about 40 per cent of this series. Thirteen per cent of these cases received Darrow's solution intravenously; the remainder received potassium by mouth or subcutaneously.

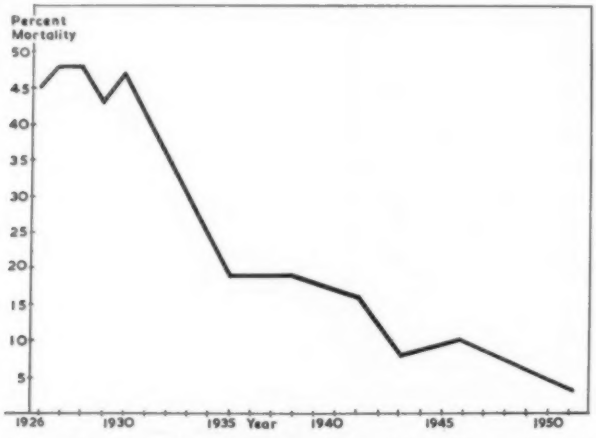

Fig. 3.—Mortality rates at the Hospital for Sick Children, 1926 to 1952.

The levels of potassium on admission ranged widely from 2.15 to 6.85 meq. per liter. This bore no relation to the sodium levels. Very low potassium levels were found in both the hypotonic and hypertonic types of values. We could also find no relationship between the duration of the diarrhea and the initial potassium values. Following rehydration there was a general lowering of the serum potassium levels. This, as expected, was more marked in patients who did not receive potassium replacement therapy.

MORTALITY

In this series of 518 infants, seventeen patients died, giving a mortality

of 3.3 per cent. Of these seventeen patients, six died within one hour of admission to hospital and received no treatment of any sort. This would give a mortality of 2.1 per cent for patients receiving treatment. Some authorities quote mortality rates only in patients admitted in a dehydrated state. This would increase our figures to 4.1 per cent for all patients and 2.6 per cent for treated patients.

The average age of infants whose illness was fatal was 3.3 months. In those cases where the infant did not die on admission, the hospital course before death was prolonged. We obtained post-mortem examinations on fourteen of these patients. Two patients were found to have evidence of infection in the mastoid air cells at post mortem, but it was not known whether this was primary, intercurrent, or terminal. In two infants there were no significant post-mortem findings, but they were found before death to have clinical evidence of hypopotassemia associated with extremely low serum potassium values below 2 meq. per liter.

It can be seen in Fig. 3 that our mortality has declined considerably through the years. This has been the experience in most other centers. Notwithstanding the importance of our progress in the understanding of this disease, we must be reasonably skeptical about therapeutic claims. The certain decrease in the virulence of this disease here in the past decade has contributed to the lowered mortality. Recognition by both parents and medical practitioners of the seriousness of gastroenteritis has enabled us to treat these children earlier than was previously possible.

SUMMARY AND CONCLUSIONS

Five hundred and eighteen cases of acute gastroenteritis in infants were admitted to the Hospital for Sick Children, Toronto, from July, 1951, through December, 1952. This disease was found in all social and financial classes of our population. It is an illness of early infancy with a peak age incidence at 2 months. The seasonal incidence, once so prominent at this center, has now largely disappeared.

The etiological agent or agents responsible for this disease are still uncertain. Only 4.4 per cent of this series were associated with a proved enteric pathogen. The relationship of parenteral infection, present in 40 per cent of the infants studied, is indefinite.

The clinical features of the dehydrated infant are presented and the methods of treatment are outlined. The value of blood or plasma transfusion and intravenous fluid therapy is well recognized. Early feedings of clear fluids are well tolerated. The gradual introduction of diluted milk feedings is advocated. Initial withdrawal of all oral feeding has not shortened the length of the vomiting or diarrhea. Antibiotic therapy is of value in the prevention of intercurrent infection and in the treatment of parenteral infections associated with the diarrhea, but has no effect upon the gastrointestinal disorder or the length of hospital stay. Opiates have no real antidiarrheal value in the treatment of this severe disease.

The mortality figures are analyzed. In this series the mortality was 3.3 per cent in all cases and 2.1 per cent in treated cases. The decline in mortality at this and other centers is noted.

The author wishes to express his appreciation to Prof. A. L. Chute, Dr. H. E. Edwards, and Dr. Harry W. Bain of the Hospital for Sick Children for their kind help and assistance in the preparation of this paper.

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SINGLE VENTRICLE WITH DIMINUTIVE OUTLET CHAMBER

Associated With Coarctation of the Aorta and Other Cardiac Anomalies

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IN REVIEWING the literature for cases of single ventricle with diminutive outlet chamber, much confusion is found in that oftentimes these cases are grouped with other anomalies, such as tricuspid atresia, under the heading of "cor triloculare."[2-10] From a functional standpoint, this is probably a reasonable classification, but from the anatomical and surgical standpoints there is no justification. Each is a distinct and separate entity with obviously different anatomical variations and surgical implications. Most cases of tricuspid atresia, aortic atresia, and single ventricle with diminutive outlet chamber are functionally and anatomically triloculate hearts. However, it may be absence of right ventricle on the one hand and left ventricle on the other that makes for absolute anatomic differences. Some in this group are operable, others inoperable. An absence of auricular or ventricular septum is not only anatomically, but functionally, a true triloculate heart and neither anomaly is operable by our current-day surgical methods. A tricuspid atresia may be helped surgically.[12] Aortic atresia is definitely inoperable, at least with surgical procedures that may alleviate symptoms in tricuspid atresia. Certain varieties of single ventricle with diminutive outlet chamber can be helped, at least theoretically, by the same surgical procedures as are now available for the tetralogy of Fallot and related anomalies. Under circumstances in which the pulmonary blood flow is reduced, such as in the case of emergence of the pulmonary artery from the diminutive chamber and the aorta from the single ventricle, operation will be of great benefit.[12] When both vessels emerge from the rudimentary chamber, the pressure in the aorta and pulmonary artery are relatively the same and surgery is obviously of little or no value. Consequently, the statement made by Conn and associates[3] that cor triloculare "should probably be ruled out when surgical treatment is considered" is without much meaning, unless one classifies only cases of absent auricular septum (cor triloculare biventriculare) or ventricular septum (cor triloculare biatriatum) under this heading. Their report lists four new cases, all biventriculare, but their discussion includes several other varieties of triloculate heart.

Taussig[11] has concisely outlined the typical findings of single ventricle with diminutive outlet chamber. Although there are three distinct types, one feature common to all three is the presence of a single ventricle into which the mitral and tricuspid valves open and a diminutive outlet chamber which arises from the single ventricle and from which one or both vessels

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arise. The three varieties include (1) the emergence of both great vessels from the diminutive chamber,[11] (2) the origin of the pulmonary artery from the diminutive chamber and the aorta from a single ventricle,[1, 11] and (3) the pulmonary artery arising from the common ventricle and the aorta from the primitive chamber.[4]

The course of the circulation is such that the blood from both auricles is emptied into the single ventricle and then flows through the diminutive outlet chamber. The vessel arising from the common ventricle is usually larger than the one emerging from the primitive chamber. This indirectly determines the degree of cyanosis. If the pulmonary artery arises from the single ventricle and thus is of larger caliber than the aorta, cyanosis will be minimal; conversely, if the pulmonary artery arises from the diminutive chamber and is therefore small, the pulmonary circulation will be much reduced and cyanosis will be more intense.

Diagnosis of this malformation is made primarily from x-ray findings. Since the diminutive outlet chamber is located in the position usually occupied by the pulmonary conus, a "full conus" will be found in the anteroposterior view. The prominent conus, usually being an index of right ventricular enlargement or more specifically right ventricular hypertension, would suggest that this situation exists from the anteroposterior view alone. However, in the left anterior oblique position, no such evidence of right ventricular hypertrophy is present, there being little or no anterior projection beyond the border of the aorta. This combination of findings should suggest, therefore, that in the absence of right ventricular hypertrophy the "conus" found in the anteroposterior view is not conus at all, but rather a diminutive outlet chamber.

In the case reported here the findings were suggestive of single ventricle with diminutive outlet chamber, but the associated anomalies were in no way suspected during life.

CASE REPORT

J. C., a 1-month-old white male infant, was admitted to St. Joseph's Hospital on July 25, 1952, because of cyanosis of several hours' duration. His birth was reported as spontaneous, at term, and there was no cyanosis noted by the attending physician. Birth weight was 9 pounds 12 ounces. There was no history of any maternal infectious disease during pregnancy, nor was there any family history of congenital heart disease.

Shortly after birth, the mother noted that the child's color was poor and his respirations were irregular, although no definite cyanosis was noted at that time. However, the mother stated that the baby turned slightly blue after crying or nursing, but this persisted for only a few minutes. The patient was on an evaporated milk formula, but nursed poorly until admission to the hospital. There was no history of any illness prior to admission.

On the morning of the twenty-fifth day of life the patient was noted to have his first attack of definite cyanosis and moderate dyspnea. At this time cyanosis was limited to his hands and face. This attack lasted approximately five minutes, following which his color and respirations improved. Another similar attack occurred shortly thereafter and he was taken to a local physician who attributed this cyanosis to "heart trouble" and advised hospitalization. En route to the hospital the patient had a severe cyanotic attack which lasted for forty minutes,

involving the entire body and during which he appeared to have stopped breathing several times. When seen at the hospital the patient was slightly cyanotic and having grunting type respirations. He suffered another mild attack while being taken from the Emergency Room to the Pediatric Ward.

Fig. 1.—Anteroposterior view. Demonstrates generalized cardiac enlargement, fullness of the "pulmonary conus," and an increase in bronchovascular markings.

Physical examination on admission revealed generalized, mild cyanosis and rapid, irregular respirations. Pulse was 156 per minute, temperature 99.8° F. rectally, and respirations 55 per minute. Examination of the lungs revealed decreased breath sounds and slight dullness in the right base. Examination of the heart revealed a moderate enlargement to percussion and a short, systolic precordial murmur. The liver edge was palpable one fingerbreadth below the right costal margin. No edema was noted and the remainder of the physical examination was essentially negative.

Admission laboratory studies showed a hemoglobin of 15.1 grams (103 per cent), red blood count of 5.66 million, and a white blood count of 10,150 with a normal differential.

X-ray of the chest revealed marked enlargement of the heart to both right and left and considerable fullness in the region of the pulmonary conus in the anteroposterior position (Fig. 1). The bronchovascular markings appeared to be increased. In the left anterior oblique view (Fig. 2) there

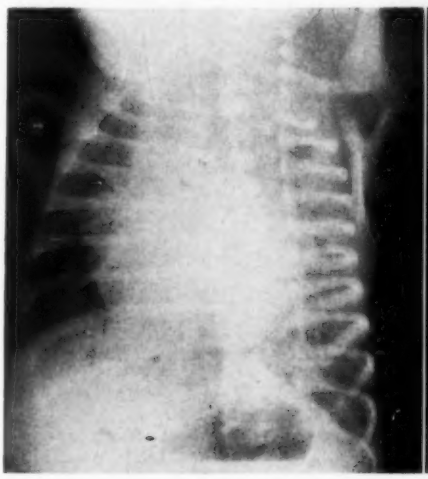

Fig. 2.

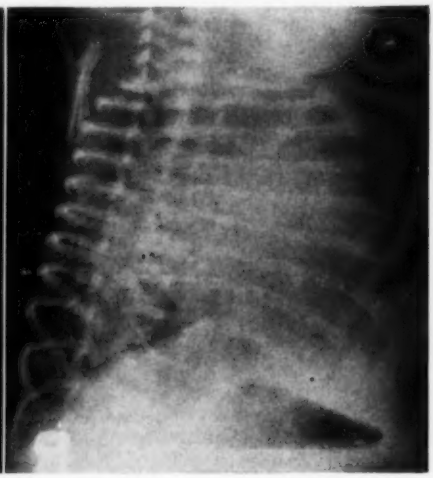

Fig. 3.

Fig. 2.—Left anterior oblique view. Note marked overlapping of the spine by left (?) ventricle and the minimal projection of the right (?) ventricle anterior to the aorta.

Fig. 3.—Right anterior oblique view. The enlargement of the right auricle is not as clearly demonstrated here as on the original films.

was considerable overlapping of the spine by the left ventricle and only minimal extension of the right ventricle anteriorly. In the right anterior oblique view (Fig. 3) the right auricle appeared enlarged.

heart was semihorizontal and the unipolar precordial leads showed primarily left ventricular activity with little or no right ventricular influence. A repeat electrocardiogram on Aug. 12, 1952, showed virtually the same

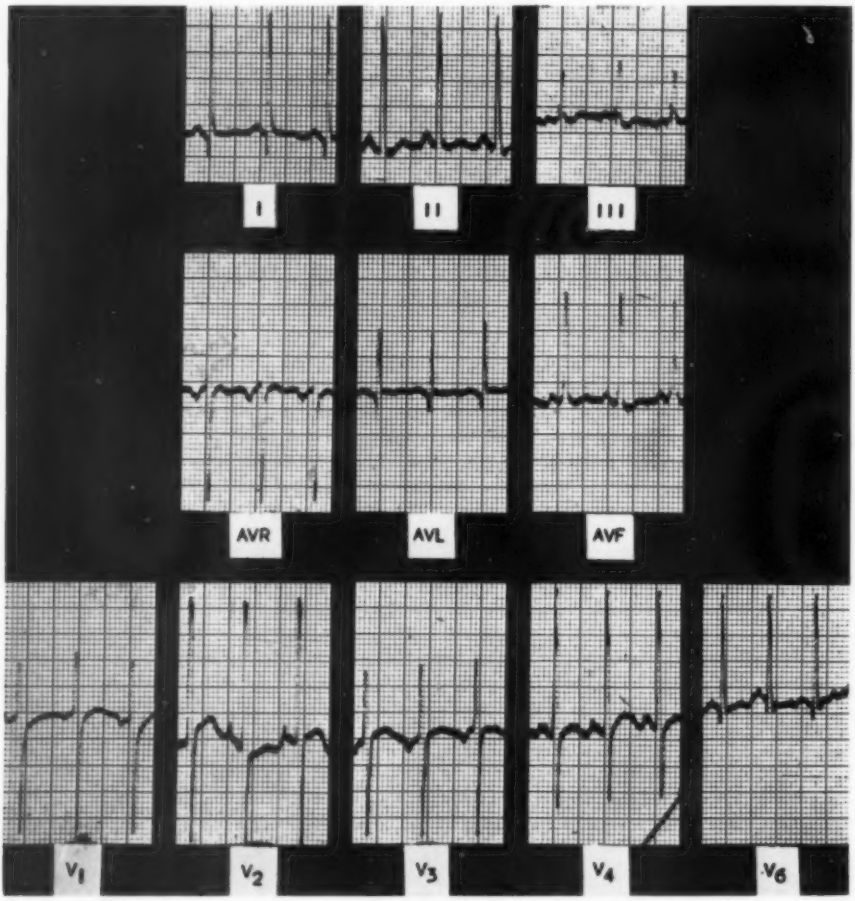

Fig. 4.—Electrocardiogram taken Aug. 12, 1952.

The initial electrocardiogram (July 25, 1952) showed a rate of 135 with regular sinus rhythm. The P-R interval measured 0.13 which was top normal for age and rate. There was an electrical axis of plus 42 degrees which represented a left axis for this age. The electrical position of the findings with exception of an axis of about plus 50 degrees (Fig. 4).

Hospital Course.—The patient was placed in an oxygen tent after admission. Cyanosis appeared to decrease and during the first two weeks was present only intermittently. At no time during hospitalization was cy-

anosis more than moderate. In the third week dyspnea, tachycardia, progressive enlargement of the heart and liver, and increased harshness of the murmur ensued. Because of these signs of cardiac decompensation, digitalization was begun and the infant's condition improved remarkably thereafter. He was given an initial dose of 33 mg. per kilogram of digitalis (whole leaf) during the first twenty-four hours, followed by a maintenance dose of 12.5 mg. a day. The child was removed from the oxygen tent for progressively longer periods of time with no return of cyanosis except when crying or nursing. On the twentieth hospital day the patient suddenly became apneic for a period of four to five minutes, but was resuscitated by the use of oxygen, stimulants, and artificial respiration. For the following month he was maintained in a satisfactory condition with an increased dose of digitalis to 25 mg. a day. Two days before death he began nursing poorly and vomited. There was marked sternal and subcostal retraction. His course was progressively downhill from this point, and despite stimulants and oxygen the patient died on Sept. 1, 1952.

During the hospital stay two attempts at arterial oxygen saturation were performed, but in both instances venous blood was obtained. Subsequent laboratory studies, including x-ray and electrocardiogram, showed no essential change from the original findings, other than a progressive anemia to a count of 3.48 million red cells and a hemoglobin of 9.3 grams (63 per cent) two days before death.

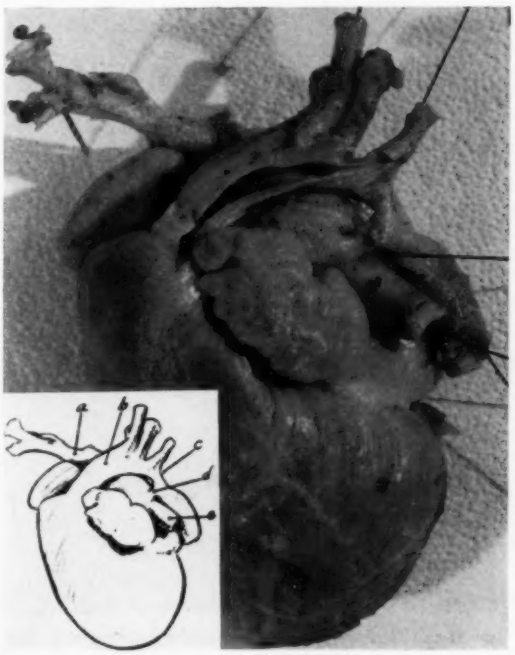

Fig. 5.—Photograph of exterior of heart. *a*, "Single" pulmonary vein. *b*, Ascending aorta. *c*, Area of coarctation. *d*, Ductus arteriosus. *e*, Left main pulmonary branch. The vessels between *b* and *c* represent the normal brachiocephalic vessels coming off the aorta as the innominate, left carotid and left subclavian arteries.

Autopsy Findings.—Although chief interest was centered about the heart, the following were general findings

noted: The body was that of a well-developed, fairly well-nourished, white, male infant measuring 22¼ inches (56 cm.) in length and weighing 8 pounds 12 ounces (3,977 grams). The skin and mucous membranes were quite pale. The scrotum was swollen and there was mild pedal edema. The abdomen was distended and the abdominal cavity contained about 300 c.c. of clear, yellow fluid. The lungs showed edema and passive congestion.

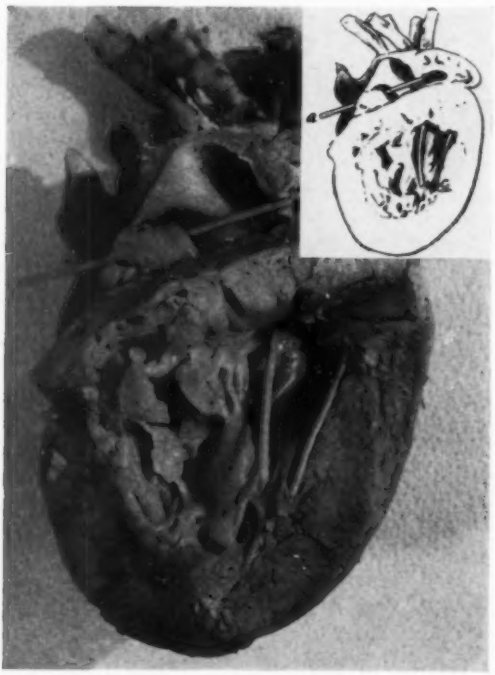

Fig. 6.—Photograph of the interior of heart. *a*, Straw passing through the patent foramen ovale from the right auricle to the left auricle. *b*, Straw entering the pulmonary artery and *c*, entering the aorta. *d*, Tricuspid valve (mitral is completely atretic).

The heart was markedly enlarged, predominantly ventricular mass and right auricle. Examination of the great vessels in situ revealed some deviation from the normal. The venae cavae emptied into the right auricle in the normal fashion, but there was only a single pulmonary vein entering the left auricle (Fig. 5). Further investigation, however, showed that there were the usual two veins from each lung, the four then uniting into a single pulmonary vein some distance from the left auricle. The aorta was found to leave the heart anterior to the pulmonary artery which indicated a transposition of the great vessels. The pulmonary artery appeared to be larger than the aorta.

The left auricle was rather small in volume, but the wall was somewhat thicker than that of the right auricle, measuring 4 to 6 mm. and 3 to 4 mm., respectively. There was a patent foramen ovale measuring 1.2 cm. in diameter. The mitral valve was completely atretic and the only exit from the left auricle was via the foramen ovale which had no membranous covering. The tricuspid valve was completely normal and opened into a large, single ventricle from which arose a diminu-

tive outlet chamber. The wall of the single ventricle was quite hypertrophied, measuring from 13 to 15 mm. in thickness. The pulmonary artery arose from the single ventricle and the aorta from the rudimentary chamber (Fig. 6). The pulmonary artery measured 13 mm. in diameter and the aorta measured 8 mm. in diameter, both measurements being taken above the heart. The valves of each vessel were competent. The brachiocephalic vessels were normal in size and position. Just distal to the point of exit of the left subclavian artery the aorta narrowed abruptly to a diameter of 3 mm. and became wider again proximal to the point of entrance of the ductus. The ductus measured 5 mm. in diameter externally, but only 2 mm. internally. The lumen was quite irregular and was thought to be undergoing obliteration.

The remaining organs were essentially normal with the exception of evidence of generalized, acute passive congestion.

DISCUSSION

The fundamental anomaly in this case was the single ventricle with diminutive outlet chamber. The fact that the pulmonary artery arose from the common ventricle precluded the possibility of successful surgical intervention since the pulmonary blood flow apparently exceeded the systemic blood flow by a considerable degree. Blood entered the right auricle by way of the superior and inferior venae cavae. It passed then through a normal tricuspid valve to a single ventricle from which the majority of blood entered the pulmonary artery and the remainder entered the diminutive chamber and thence traveled to the aorta.

The blood traversing the pulmonary artery entered the lungs where it is assumed normal oxygen-carbon dioxide exchange took place and aerated blood returned to the left ventricle. From here, on account of the mitral atresia, all the blood passed through the patent foramen ovale and mixed with the blood returning from the systemic circulation via the venae cavae.

The blood entering the aorta apparently met with some obstruction because of the coarctation. Since the ductus entered the aorta distal to the coarctation it is assumed that blood flow through the ductus was from the pulmonary artery to the aorta and thus maintained a fair degree of systemic flow. It appeared that the obliteration of the ductus was beginning to interfere with this systemic blood flow and consequently may have contributed in some degree to the death of the patient.

The presence of a coarctation was not suspected. Although the blood pressure was not taken there seemed no necessity, at the time, in view of the fact that the femoral pulse appeared adequate on palpation. On each of the two attempts at drawing blood from the femoral artery for oxygen saturation, there was no suggestion that femoral blood flow was in any way reduced.

The patent ductus gave no evidence of its presence by way of a typical continuous murmur. This is not especially uncommon since a concomitant ductus, whether compensatory or not, does not always give evidence of its presence. Furthermore, there was some suggestion at autopsy that the ductus was undergoing obliteration.

The absence of a mitral valve complicated the situation to only a minor degree, since all the blood from the two auricles eventually pooled in the

common ventricle. There was, however, some apparent increased burden on the heart in getting blood from the left auricle to the common ventricle through the devious route necessitated by the mitral atresia.

The single pulmonary vein was of no significance except from an academic standpoint since there was no obstruction to return of blood from the lungs.

The multiplicity of anomalies associated with this case is not particularly unusual.[2, 4-10, 13] The case is presented here more from the standpoint of clarifying a single ventricle with diminutive outlet chamber in the literature than presenting one associated with other abnormalities.

SUMMARY

1. A case of single ventricle with diminutive outlet chamber associated with multiple, additional anomalies is presented.

2. Confusion which exists in the literature in incorporating these and other distinct anomalies under the heading of cor triloculare is cited.

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THERAPEUTIC DOSES OF GAMMA GLOBULIN IN THE TREATMENT OF MEASLES ENCEPHALITIS AND ENCEPHALOMYELITIS

II. Cerebrospinal Fluid and Serum Studies in Measles Encephalitis and Encephalomyelitis With Special Reference to Phosphorus, Cholesterol, and Magnesium

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DURING the 1952 epidemic of measles a number of our patients with measles encephalitis and encephalomyelitis were treated with large doses of gamma globulin and adjunctive therapy in the acute phase. The results of this therapy are given in the first paper.†

Especially interesting were some of the serum and cerebrospinal fluid findings in these patients; we found elevated inorganic phosphorus and cholesterol cerebrospinal fluid levels together with hypophosphatemia. Our findings will be discussed in relation to the pathologic changes in measles encephalitis and encephalomyelitis. Data on serum and cerebrospinal fluid inorganic phosphorus and on cerebrospinal fluid cholesterol in measles encephalitis and encephalomyelitis have not been previously reported. The role of magnesium in the central nervous system complications of measles will also be discussed. These findings have been applied to the therapy in the acute stage of measles encephalitis and encephalomyelitis presented in the first paper.

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Furthermore, an unusually high percentage of pathologic cerebrospinal fluids were found in the forty-one cases herein reported as compared to reports in the literature; 94 per cent of the fluids examined revealed pleocytosis, and 92 per cent, elevated total protein.

The data on the sera and cerebrospinal fluids are tabulated in Tables I and II. The case numbers in Tables I, II and III in this paper correspond with the clinical histories reported in the first paper.

BLOOD FINDINGS IN MEASLES ENCEPHALITIS AND ENCEPHALOMYELITIS

White cell counts of the blood were made in thirty-five out of thirty-six cases (97 per cent) in the acute phase (Table I). Of these, twenty-four (69 per cent) had an elevated white cell count with a range of 10,100 to 35,000 per cubic millimeter and eleven (31 per cent) had a normal count with a range of 6,100 to 9,500 per cubic millimeter. Of the twenty-four patients whose white count was elevated, ten had a relative granulocytosis, six had a relative lymphocytosis, and eight had a normal differential count. The distribution of the eleven normal white cell counts was as follows: three relative granulocytosis, seven relative lymphocytosis, and one normal differential

TABLE I. BLOOD FINDINGS IN CASES OF MEASLES ENCEPHALITIS AND ENCEPHALOMYELITIS

CASE NO.	MONTH AND YEAR ADMITTED	AGE YEARS	HOSPITAL DAY	WBC PER C.MM.	NEUTROPHILS %	LYMPHOCYTES %	EOSINOPHILS %	BASOPHILS %	SUGAR MG. %	UREA N MG. %	TOTAL PROTEIN GM. %	ALBUMIN GM. %	GLOBULIN GM. %	TOTAL CHOLESTEROL MG. %	TOTAL Cl MG. %	CO_2 VOL. %	Na MG. %	K MG. %	Ca MG. %	P MG. %
1.	6-49	10	Adm.	34,500	90	10	0	0								52				
			6	20,500	68	32	0	0												
			14	13,900	68	32	0	0												
2.	7-49	7½	Adm.	11,900	60	40	0	0												
3.	5-50	11	3	8,200	50	50	0	0							350	60				
4.	6-50	8	Adm.	10,100	56	44	0	0												
5.	6-50	2	None																	
6.	7-51	5	Adm.	7,100	55	44	0	1		12.5						49				
7.	7-51	5	Adm.	8,300	72	28	0	0			6.0	4.0	2.0			52				
8.	8-51	6	Adm.	11,600	72	28	0	0												
9.	8-51	6	Adm.	8,900	84	14	2	0												
			6	7,600	66	28	6	0												
10.	12-51	3½	None																	
11.	1-52	1¼	Adm.	7,800	44	56	0	0												
12.	2-52	10/12	Adm.	6,500	31	68	0	1												
			5	8,800	14	82	4	0												
			26	7,300	26	62	12	0												
13.	2-52	7	4	11,400	66	34	0	0												
14.	5-52	5	Adm.	7,800	62	38	0	0												
			2						100	9.4				151		50	308		10.8	2.6
15.	1-52	3	Adm.	16,700	67	32	0	1												
16.	1-52	8	Adm.	6,100	75	25	0	0									320	20.6		
			6	12,100	80	20	0	0							362		331	18.1		
17.	2-52	4	6*	12,100	74	26	0	0												
18.	2-52	6	None																	
19.	2-52	13	4	10,700	44	56	0	0												
20.	3-52	9	2	15,700	66	34	0	0												
			6	12,100	52	46	2	0												
21.	3-52	7	Adm.	13,800	86	14	0	0												
			6	3,100	40	60	0	0												
22.	3-52	8	Adm.	11,000	70	30	0	0												
23.	3-52	5	None																	
24.	3-52	2½	Adm.	11,900	46	54	0	0												

25.	3-52	6	Adm.	6,700	76	24	0	0										
			10	11,100	74	26	0	0										
			25								6.0	4.1	1.9	260				
			62								7.1	5.0	2.1					
26.	4-52	7	Adm.	13,800	82	18	0	0										
27.	8-50	6	None															
28.	2-52	5½	6	8,600	62	38	0	0										
29.	4-52	7	Adm.	26,200	75	25	0	0										
			3	7,800	58	42	0	0		17.0	6.5	4.0	2.5	240	54		9.8	
30.	4-52	8	Adm.	14,500	72	28	0	0	100	10.6	5.9	4.6	1.3	157	72			3.7
			9	4,200	40	58	2	0										
31.	5-52	13	Adm.	8,100	44	56	0	0	140	9.0	6.9	3.6	3.3		56		9.8	3.2
32.	4-52	4	4†	30,000	88	12	0	0		12.0	5.4	3.3	2.1	220	49		8.6	4.7
			7	8,500	58	38	4	0										
33.	4-52	5½	2	17,400	66	34	0	0						272	45	335	20.0	
			4	15,500	66	34	0	0										
34.	4-52	5	Adm.	15,000	64	36	0	0										
			2	7,700	50	48	0	2						275			11.3	2.9
35.	4-52	10½	Adm.	11,900	74	26	0	0	91	9.5				206	53	338	9.8	5.1
			2	13,700	49	50	0	1										
			16	10,800	50	48	2	0										
36.	5-52	6½	Adm.	14,800	76	24	0	0	121	9.5				227		292	10.1	3.5
			6	8,600	54	44	2	0										
37.	5-52	3½	Adm.	6,800	58	40	2	0	90	12.0	6.2	3.5	2.7	164		278	14.0	11.0	2.5
38.	5-52	9	Adm.	21,800	84	16	0	0	98	11.1	6.9	3.8	3.1	244		300	15.0	9.2	2.5
			3	37,800	77	22	0	1										
			9	19,900	84	14	2	0										
			13	14,900	70	29	1	0										
39.	5-52	5	Adm.	15,700	46	54	0	0	138	9.0				258	38	308	10.7	2.8
			2	6,100	42	58	0	0										
			10	5,100	50	50	0	0										
40.	5-52	10	Adm.	22,800	84	16	0	0										
			4	4,700	34	65	1	0										
41.	5-52	7	Adm.	9,450	75	24	1	0	124	10.0				252	44	300	23.0	11.5	2.5
			18	13,000	78	20	2	0						141					2.1
			26	7,200	66	30	4	0	107					175					4.8
			33								7.6			205	65				5.1

*Onset of encephalitis.
†Onset of encephalomyelitis.

TABLE II. CEREBROSPINAL FLUID DATA IN MEASLES ENCEPHALITIS AND ENCEPHALOMYELITIS

CASE NO.	MONTH AND YEAR ADMITTED	AGE YEARS	HOSPITAL DAY	CELLS PER C.MM.	TOTAL PROTEIN MG. PER 100 ML.	GLOBULIN	SUGAR MG. PER 100 ML.	CHLORIDE MG. PER 100 ML.	TOTAL CHOLESTEROL MG. PER 100 ML.	INORGANIC PHOSPHORUS MG. PER 100 ML.
1.	6-49	10	Adm.	340	132	tr.	82			
			1	124	138	ft. tr.	81			
			35	0	17	neg.	47			
2.	7-49	7½	Adm.	226	56	v. ft. tr.	78			
3.	5-50	11	Adm.	30	74	ft. tr.	82			
			10	0	57	v. ft. tr.	74			
4.	6-50	8	Adm.	620	96	tr.	66			
5.	6-50	2	Adm.	40	44	v. ft. tr.	68			
6.	7-51	5	Adm.	120	120	ft. tr.	65			
7.	7-51	5	Adm.	70	113	ft. tr.	111			
8.	8-51	6	Adm.	38	71	ft. tr.	79			
9.	8-51	6	Adm.	150	56	ft. tr.	76			
10.	12-51	3½	Adm.	207	—	ft. tr.	77			
11.	1-52	1¼	None	—	—	—	—			
12.	2-52	10/12	Adm.	0	14	neg.	71			
			26	2	44	neg.	64			
13.	2-52	7	Adm.	75	33	neg.	110			
14.	5-52	5	Adm.	57	47	v. ft. tr.	60	650	neg.	1.9
			5	22	112	ft. tr.	59			
15.	1-52	3	Adm.	79	55	v. ft. tr.	152			
16.	1-52	8	Adm.	—	42	neg.	190			
17.	2-52	4	6*	104	44	v. ft. tr.	102			
18.	2-52	6	Adm.		106	ft. tr.	95			
19.	2-52	13	Adm.	93	150	ft. tr.	78			
			5	11	46	neg.	74			
20.	3-52	9	Adm.	—	78	tr.	88			
			6	8	54	v. ft. tr.	68			
21.	3-52	7	None	—	—	—	—			
22.	3-52	8	Adm.	614	99	ft. tr.	73			
			8	6	40	neg.	69			
23.	3-52	5	Adm.	700	130	ft. tr.	82			
24.	3-52	2½	Adm.	53	46	neg.	79			
			8	0	27	neg.	76			
25.	3-52	6	Adm.	172	63	v. ft. tr.	73			
			10	13	44	neg.	77			
			17	4	57	v. ft. tr.	78			
			55	6	45	v. ft. tr.	73			
26.	4-52	7	Adm.	—	58	v. ft. tr.	78			
			1	4	51	v. ft. tr.	135			
27.	8-50	6	Adm.	70	55	ft. tr.	70			
28.	2-52	5½	Adm.	78	110	ft. tr.	141			
29.	4-52	7	Adm.	30	102	tr.	141			
			5	18	81	ft. tr.	74			
30.	4-52	8	Adm.	220	73	v. ft. tr.	73			
31.	4-52	13	Adm.	387	72	ft. tr.	78	635	neg.	2.6
			9	0	38	neg.	74			
32.	4-52	4	4†	66	30	neg.	101			
			11	3	20	neg.	70	660	neg.	1.7
33.	4-52	5½	Adm.	40	54	v. ft. tr.	76	660		1.7
			7	0	42	neg.	74	660	neg.	1.7
34.	4-52	5	Adm.	660	65	ft. tr.	64	640		1.6
			8	9	72	ft. tr.	114		82	2.4
			14	2	36	neg.	70	600	neg.	2.0
35.	4-52	10½	Adm.	188	132	ft. tr.	69	658	85	2.2
			7	9	66	v. ft. tr.	74	660	neg.	2.0
36.	5-52	6½	Adm.	1080	78	ft. tr.	88	642	neg.	2.1
			5	122	46	neg.	76	650	neg.	1.7
			12	25	33	neg.	75	625	neg.	2.2

*Onset of encephalitis.
†Onset of encephalomyelitis.

TABLE II—CONT'D

CASE NO.	MONTH AND YEAR ADMITTED	AGE YEARS	HOSPITAL DAY	CELLS PER C.MM.	TOTAL PROTEIN MG. PER 100 ML.	GLOBULIN	SUGAR MG. PER 100 ML.	CHLORIDE MG. PER 100 ML.	TOTAL CHOLESTEROL MG. PER 100 ML.	INORGANIC PHOSPHORUS MG. PER 100 ML.
37.	5-52	3½	Adm.	22	—	tr.	90			
			5	—	42	v. ft. tr.	76	660	neg.	2.0
38.	5-52	9	Adm.	388	102	tr.	77	650	neg.	1.6
			10	4	33	neg.	62	625	neg.	2.0
39.	5-52	5	Adm.	33	45	v. ft. tr.	88		75	2.0
			8	8	43	v. ft. tr.	76	660	neg.	4.1
			12	4	30	neg.	60	660	neg.	2.0
40.	5-52	10	Adm.	194	65	ft. tr.	76		neg.	1.9
			5	30	49	v. ft. tr.	59		neg.	1.6
41.	5-52	7	Adm.	708	74	ft. tr.	71	650	neg.	1.3
			7	0	48	v. ft. tr.	72			
			13	7	46	neg.	79	560		2.2
			18	0	40					1.4
			26	2	15	neg.	73	650		1.6
			54	0	34	neg.	88			

count. The characteristic white cell count in measles encephalitis and encephalomyelitis is that of an acute infection with an elevation of the white cell count with a tendency toward a relative granulocytosis. Where the white cell count remains normal a relative lymphocytosis predominates. No leukopenia was found in any of our cases. This is in contrast to the typical uncomplicated measles cases which generally show a leukopenia with a relative lymphocytosis.

Acidosis and changes in the electrolyte balance were among the physiologic factors considered in these cases. The blood chemistries, glucose, total protein, albumin-globulin ratio, chloride, urea nitrogen, sodium, potassium, total cholesterol, and calcium were within normal limits (Table I). Of the determinations done on admission, several values reflected the dehydrated, fasting, convulsive, or encephalitic state of the patient: two sugars were on the upper limit of normal, 138 and 140 mg. per cent; three of nine total cholesterols were above 250 mg. per cent; the carbon dioxide content in seven of nine patients was low, ranging between 38 and 53 vol. per cent; and two potassiums were on the lower limit of normal, 14 and 15 mg. per cent. All cultures were sterile. The serum inorganic phosphorus findings are discussed separately.

CEREBROSPINAL FLUID FINDINGS IN MEASLES ENCEPHALITIS AND ENCEPHALOMYELITIS

Thirty-nine out of forty-one cases reported in this paper (Table II) had lumbar punctures, and cell counts were done on thirty-six of these; thirty-four (94 per cent) varied from 22 to 1,080 cells per cubic millimeter and only two (6 per cent) had normal counts: 0 and 4 cells per cubic millimeter. There were six patients (17 per cent) with cell counts over 600 per cubic millimeter. Lymphocytes predominated in thirty-three (92 per cent) cases and constituted 84 per cent or more of the cells present; in the other three cases (8 per cent) granulocytes predominated.

The total protein was determined in thirty-seven out of the forty-one cases. In thirty-four (92 per cent) cases it ranged between 42 and 150 mg. per 100 ml. and in only three cases (8 per cent) was the total protein within normal limits (14 to 33 mg. per 100 ml.).

In thirty-three cases both cell count and total protein were done. Both the cell count and the total protein were increased in the acute phase but, as was expected, the degree of increase in the total protein did not parallel the degree of increase in the cell count.

The lowest sugar found in the acute stage was 60 mg. per 100 ml. and the highest was 190 mg. per 100 ml. Ten patients had sugar in excess of 100 mg. per 100 ml.

Thus the cerebrospinal fluid analyses show definite pathologic changes in over 90 per cent of our cases. The cerebrospinal fluid inorganic phosphorus and cholesterol findings are discussed elsewhere.

All the direct smears were negative for organisms and all the cultures proved sterile.

We did not consider the cerebrospinal fluid pressure important because we found, in accord with Merritt and Fremont-Smith[1] and Spurling[2] that most children (excluding those in coma or in stupor) strained, cried, and struggled during the tap, causing an increase in pressure. Merritt and Fremont-Smith have reported that in a series where punctures were done in quiet and relaxed patients the cerebrospinal fluid pressure was normal in 82 per cent of patients with encephalitis and only slightly elevated in the remainder.

PHOSPHORUS IN MEASLES ENCEPHALITIS AND ENCEPHALOMYELITIS

Phosphorus is one of the largest mineral constituents of the brain in both the gray and the white matter.[3] It is also an important and interesting mineral metabolite of the central nervous system.

In acute infections of the central nervous system as measles encephalitis and encephalomyelitis one would expect the normal histology of the central nervous system to be altered and that evidence of such unbalance would be found in many constituents, including phosphorus, in the cerebrospinal fluid. As a matter of fact, many of the cerebrospinal fluid inorganic phosphorus values were found to be elevated in the twelve patients in whom this constituent was determined (by the modified method of Fiske and SubbaRow recently described by Odessky, Rosenblatt, Bedo, and Landau[4]).

This high cerebrospinal fluid inorganic phosphorus can be correlated with the pathologic findings in measles encephalitis and encephalomyelitis. It has been found, microscopically, that diffuse perivascular demyelination (destruction of phosphorus-containing tissue) and varying quantities of fat both in the phagocytic glia cells and in the surrounding tissue are almost always present. This observation has been made over and over again in the pathologic studies of measles encephalitis and encephalomyelitis reported by Wohlwill,[5] Creutzfeld,[7] Siegmund,[8] Sjövall,[9] Greenfield,[10] Zimmerman and Yannet,[11] Ferraro and Scheffer,[12] Finley,[13] Moore and McCordock,[14] Malamud,[15] Ziskind and Schattenberg,[16] Malamud,[17] Litvak, Sands, and Gibel,[18] Reisman and Rosen,[19] Litvak,[20] Saw-

chuk and associates,[21] Applebaum, Dolgopol, and Dolgin,[22] and Hoffman,[23] and of virus diseases in general reported by Baker.[24]

Myelinated fibers constitute the major part of the white matter in the normal brain and spinal cord. This is the portion of the central nervous system most affected in measles encephalitis and encephalomyelitis. The myelin sheath which surrounds the axis cylinder is made up largely of free cholesterol, cerebrosides, and the phospholipid sphingomyelin. In the infections of measles encephalitis and encephalomyelitis, fragments of myelin were observed around the blood vessels intracellularly and extracellularly.[5, 11, 15] That these fragments are transported into the Virchow-Robin spaces, was demonstrated by the presence of clusters of gitter cells within these spaces; hence, the disintegration of the damaged portions of the myelin sheaths would give rise, in some degree, to the elevation of the cerebrospinal fluid inorganic phosphorus and this was amply demonstrated in the studies in the *acute* phase when it was found that many patients had an increase in cerebrospinal fluid inorganic phosphorus (Table II). Eight out of twelve patients showed a cerebrospinal fluid inorganic phosphorus of 2.0, 2.2, 2.4, 2.6, and 4.1 mg. per 100 ml. in the acute phase. Two other patients had an inorganic phosphorus of 1.9 mg. per 100 ml. which is definitely on the upper limit of normal. The two remaining patients had inorganic phosphorus levels of 1.7 mg. per cent which is considered normal but because spinal punctures are done only for diagnostic purposes the possibility exists that we might not have performed the punctures at the time when the cerebrospinal fluid inorganic phosphorus was elevated.

The normal average cerebrospinal fluid value for inorganic phosphorus in children, aged 16 months to 14 years is 1.4 mg. per 100 ml.[4] (Table III). An average normal cerebrospinal fluid inorganic phosphorus of 1.63 mg. per cent for six children below 14 years of age was found by Cohen[25] (criterion for normalcy not stated). Wilcox, Lyttle, and Hearn[27] found an average value of 1.7 mg. inorganic phosphorus per 100 ml. in cerebrospinal fluids from children with degenerative diseases of the central nervous system. An average value of 1.7 mg. per 100 ml. cerebrospinal fluid inorganic phosphorus was found in fourteen infants by Okuda.[32] Few normal values of inorganic phosphorus of the cerebrospinal fluid obtained from children are found in the literature because lumbar punctures are never done routinely. Cohen,[25] who has made extensive determinations of cerebrospinal fluid inorganic phosphorus in various diseases, declared that he considered a figure of 2.0 mg. per 100 ml. as *abnormally* high and the work of Odessky and associates[4] bears out this statement. In addition it was emphasized by Merritt and Fremont-Smith[1] that the phosphorus content of the cerebrospinal fluid is remarkably constant. Our measles cases with encephalitis or encephalomyelitis showed no constancy but, instead, varied with the degree of demyelination and destruction of nerve tissue, and the derangement of the normal phosphorus metabolism of the central nervous system. The average increase in our measles cases with complications of the central nervous system was 43 per cent over our average normal children,

TABLE III. RATIO OF CEREBROSPINAL FLUID INORGANIC PHOSPHORUS TO SERUM INORGANIC PHOSPHORUS CALCULATED FROM OUR CASES AND COMPARED TO RANGE OF NORMAL AVERAGE VALUES IN CHILDREN OBTAINED FROM THE LITERATURE

CASE	AGE YEARS	HOSPITAL DAY*	CEREBROSPINAL FLUID INORGANIC PHOSPHORUS MG. PER 100 ML.	SERUM INORGANIC PHOSPHORUS MG. PER 100 ML.	RATIO $\frac{\text{CSF INORGANIC PHOSPHORUS}}{\text{SERUM INORGANIC PHOSPHORUS}}$
14.	5	Adm.	1.9	2.6	0.73
31.	13	Adm.	2.6	3.2	0.81
34.	5	Adm.	1.6	2.9	0.55
35.	10½	Adm.	2.2	5.1	0.43
36.	6½	Adm.	2.1	3.5	0.60
38.	9	Adm.	1.6	2.5	0.64
39.	5	Adm.	2.0	2.8	0.71
41.	**7**	Adm.	1.3	2.5	0.52
		18	1.4	2.1	0.67
		26	1.6	4.8	0.33†
Source					
Odessky, and associates[4]	Under 14		1.4		
Cohen[25]	Under 14		1.6		
Pineus and Kramer[26]	(Adults)		1.2		
Wilcox, Lyttle, and Hearn[27]	Under 14		1.7		
Scholberg and Goodall[28]	(Adults)		1.4		
Steiner[29]	--------		1.5		
Lierle and Sage[30]	--------		1.7		
Fremont-Smith and associates[31]	(Adults)		1.5		
Okuda[32]	Infants		1.7		
Cohn, Kaplan, and Levinson[33]	--------		1.4		
Howland and Kramer[34]	Under 14			4.5-6.8	
Bullock[35]	Under 14			4.0-6.7	
Robinson[36]	Children			4.5-5.5	
Range of average normal values					$\frac{1.2}{6.8} - \frac{2.0}{4.0}$
			1.2-2.0	6.8-4.0	0.18 - 0.50

*The cerebrospinal fluid and serum specimens were taken on the same day.
†The lumbar puncture at this time and also one week later showed normal cerebrospinal fluid values.

a relatively large and pathognomonic increase in the cerebrospinal fluid for the mineral phosphorus. The mean cerebrospinal fluid inorganic phosphorus level for the twenty-three fluids from twelve patients is 2.02 mg. per cent. The standard error of the mean is ±0.11. The difference between the means of these measles encephalitis and encephalomyelitis cases, 2.02 mg. per cent, and our control patients, 1.4 mg. per cent, is 3.5 times the sum of the respective standard errors, ±0.11 and ±0.06, indicating that our data are statistically significant.

Concomitant with the increase in the cerebrospinal fluid inorganic phosphorus there should be found an increase in the phosphate-splitting enzymes. While no work has been reported in measles with central nervous system complications, Demuth[37] and Kaplan and co-workers[38] did find that cerebrospinal fluid phosphatase was increased in pathologic conditions, and Hicks[39] found that a slight increase in phosphatase activity in mechanically injured brain tissue occurred.

The laboratory studies, furthermore, show that an abnormal ratio of

$$\frac{\text{CSF inorganic phosphorus}}{\text{serum inorganic phosphorus}}$$

(Table III) exists in measles encephalitis and encephalomyelitis. Using the lower and upper extremes of normal values (Table III) for children under 14 years of age for serum inorganic phosphorus (4.0 to 6.8 mg. per 100 ml.) and for cerebrospinal fluid inorganic phosphorus (1.2 to 2.0 mg. per 100 ml.), the normal range of the ratio of cerebrospinal fluid inorganic phosphorus to serum inorganic phosphorus was obtained: $\frac{1.2}{6.8} = 0.18$; $\frac{2.0}{4.0} = 0.50$; therefore, the range of the normal ratio is 0.18 to 0.50. For comparison, the average ratio for adults in nonsuppurative diseases has been stated to be 0.38.[40]

From Table III it can be seen that seven out of eight children had ratios above the upper limit of normal (0.52 to 0.81) and the eighth child had an elevated ratio (0.43). This increase in ratio is attributable not only to the elevated cerebrospinal fluid inorganic phosphorus, generally more elevated in the *acute* stages than in the *chronic* stages of a disease, but also to the decreased serum inorganic phosphorus (Tables I and III). Of eleven children whose serum inorganic phosphorus was determined in the acute phase only one child had a completely normal serum inorganic phosphorus, and Case 30, with a serum inorganic phosphorus of 3.7 mg. per 100 ml., was on the lower limit of normal. Thus nine out of eleven children had hypophosphatemia with 2.1 to 3.5 mg. inorganic phosphorus per 100 ml. of blood. It might be considered that the hypophosphatemia, at least in part, might contribute to the lowered resistance of the child and subsequent viral invasion of the central nervous system as hypothesized in the discussion on the etiology of encephalitis and encephalomyelitis complicating measles presented in the first paper.

CHOLESTEROL AND LIPIDS IN THE CENTRAL NERVOUS SYSTEM IN MEASLES ENCEPHALITIS AND ENCEPHALOMYELITIS

Lipids and cholesterol make up the largest part of the brain substance constituting 71 per cent of the white matter of dry tissue and 44 per cent of the gray matter of dry tissue.[41]

In measles encephalitis and encephalomyelitis there is destruction of the myelinated nerve tissue (composed of cholesterol, phospholipids, and other substances) and, as a consequence, increased cholesterol and lipids in the cerebrospinal fluid might be expected to occur. In fact we found abnormally high cerebrospinal fluid total cholesterol values in several patients during the acute phase of the disease. Three of our cases (34, 35, and 39) showed cerebrospinal fluid total cholesterol levels of 82, 85, and 75 mg. per 100 ml., respectively (method of Bloor, Pelkan, and Allen[42-44]).

In normal individuals, children and adults alike, the value for cholesterol in the cerebrospinal fluid is essentially negative—less than 0.2 mg. per 100 ml. Nevertheless, in a review of the literature by Katzenelbogen,[45] it was found that from time to time cerebrospinal fluids containing considerable quantities of cholesterol have been reported, the highest figure being 72 mg. per 100 ml. in a case of dementia precox. Relatively few elevated cerebrospinal fluid cholesterols have been reported: first, because this determination is rarely carried out, and second, the work of Plaut and Rudy,[46] who injected rabbits with cholesterol suboccipitally, indicates that cholesterol in the cerebrospinal fluid recedes to normal values fairly rapidly. When sphingomyelin, lecithin, cholesterol, and whole brain lipids were injected into adult mice brains by Hicks,[47] no trace of these substances were found in the wound tracks inferring that the lipids were absorbed or carried away. Additionally, the determinations of enzymes in the cerebrospinal fluid by Kaplan and associates[38] revealed that the enzymes, lipase, amylase, esterase, phosphatase, trypsin, and tributyrinase are increased in pathologic conditions and the authors concluded that increased enzymatic activity of the pathologic cerebrospinal fluid may be due to (a) increased meningeal permeability, (b) increased number of leukocytes, or (c) destruction of brain tissue.

Evidence of the presence of lipids and cholesterol in deteriorated tissue has been encountered in microscopic sections of brain tissue from patients who died from the effects of measles encephalitis and encephalomyelitis. Fat has been observed in varying quantities, depending upon the amount of demyelination, engulfed by the mononuclear cells and lying free in the tissues. Histologic descriptions of lipids in measles encephalitis and encephalomyelitis have been reported by Wohlwill,[5] Guillery,[48] Creutzfeld,[7] Siegmund,[8] Sjövall,[9] Greenfield,[10] Zimmerman and Yannet,[11] Ferraro and Scheffer,[12] Moore and McCordock,[14] Malamud,[15] Ziskind and Schattenberg,[16] Malamud,[17] and Appelbaum, Dolgopol, and Dolgin.[22] Litvak, Sands, and Gibel[18] found fat in the pia and this was confirmed by Dr. Otto Marburg who studied, histologically, brain sections sent to him by these authors. There is an increased number of gitter cells in the white matter in measles encephalitis and encephalomyelitis, and an occasional widened perivascular space is seen crowded with gitter cells. From the Virchow-Robin spaces these scavenger cells containing cholesterol and lipids are transported into the cerebrospinal fluid whence we find abnormally high cholesterol values.

All sections obtained from autopsies from measles encephalitis and encephalomyelitis stained with scharlach red, scarlet red, Sudan III, and Sudan IV take the red stain, evincing the presence of broken-down lipid tissue, yet many of these sections containing microscopically large quantities of lipoid globules do not show a uniform distribution of the stain with these neutral dyes. Not every globule took the fat stain and some globules stained only fragmentarily, suggesting that the fat globules are not all neutral fat but are other lipids such as cerebrosides, phospholipids, and cholesterol. With the aid of the polarizing microscope, Greenfield,[10] moreover, discerned numerous colorless crystals of cholesterin in brain sections from a case of measles encephalomyelitis.

The foregoing discussion on phosphorus, cholesterol, and lipids presents histologic, chemical, and physical evidence, gathered from the cases in the literature and our patients, of the breakdown of myelin and the destruction of nerve tissue in measles encephalitis and encephalomyelitis.

MAGNESIUM IN THE CENTRAL NERVOUS SYSTEM

Considering the importance of this mineral constituent of the central nervous system very little is known about its role in encephalitis and encephalomyelitis complicating viral infections. Lewis[49] emphasized our lack of knowledge of magnesium by pointing out that the reason why this mineral is present in the fetal brain in larger amounts than in any other fetal organ is unknown. According to Alexander and Myerson,[3] who determined the minerals in normal and pathologic brain tissue by microincineration and spectroscopy, the minerals, in order of decreasing concentration, are: phosphorus, magnesium, sodium, potassium, iron, calcium, copper, and manganese. In marked contrast to the extensive literature on the electrolytes in the central nervous system, such as sodium, potassium, iron, and calcium, there is a paucity of reports on phosphorus and magnesium. One explanation for the lack of data on magnesium is that magnesium is widely distributed in foods—in nuts, grains, legumes, fish, poultry, potatoes, vegetables, meat, fruits, eggs and milk (Sherman[50]; listed in order of decreasing concentration) and is, therefore, readily and regularly available to people who *eat* a normal, varied diet although it is also regularly excreted via the feces and urine. However, in acute febrile stages of viral infections of the central nervous system where the nervous tissue suffers extensive damage and where the metabolic rate is increased considerably, the patient will be in negative magnesium balance, especially if there has been a period of anorexia and vomiting resulting in lowered food intake as is wont to occur in illness. Duckworth and Warnock[51] believe that more magnesium is excreted via sweat than is generally supposed and concluded that possibly the loss of magnesium during periods of illness may be far greater than is realized, an important point to be considered in the hyperpyrexia of central nervous system complications of measles.

Magnesium deficiency will produce symptoms and lesions referable to the central nervous system and skeletal muscles. Experimentally, Kruse, Orent, and McCollum[52, 53] reported that when rats and dogs were fed a

diet deficient in magnesium but adequate in other constituents the animals developed vasodilation and hyperirritability of the central nervous system followed by convulsions. A case of tetany due to a serum magnesium deficiency (the serum calcium was normal but the serum phosphorus was low) in a 6-year-old child was treated with magnesium sulfate by Miller[54] with subsequent disappearance of the patient's tremors. Histologically, Lowenhaupt, Schulman, and Greenberg[55] found widely distributed inflammatory, necrotic, perivascular lesions in magnesium-deficient rats. An almost constant location was the fibrous septums of skeletal muscle both that of the upper extremities and that of the diaphragm. The lesions were also observed in the central nervous system.

A review of the literature by Katzenelbogen[45] revealed that the values for magnesium in various pathologic conditions vary greatly. However, it appears to have been established that the magnesium content of the cerebrospinal fluid is greater than that of the blood by about 25 per cent. In pathologic conditions as tuberculosis and meningococcal meningitis the cerebrospinal fluid magnesium is lowered and approaches the level of magnesium in the blood.

Magnesium functions as a coenzyme in many of the enzyme systems concerned with the glycogen metabolism of the brain (in which phosphate is a substrate).[56, 57, 58, 59, 60] Finally, magnesium, a large component of skeletal muscle, exceeded in quantity by potassium, phosphorus, sodium, and chloride, must be provided when muscles are affected in central nervous system complications caused by virus.

For the foregoing reasons magnesium sulfate was injected intramuscularly on admission and in small doses daily thereafter or antacid doses of magnesium hydroxide were given daily by mouth until a balanced diet could be taken by the patient which would furnish the central nervous system with the constituents necessary for its normal metabolic maintenance and also would enable it to repair the damage inflicted by viral infections.

SUMMARY AND CONCLUSIONS

The cerebrospinal fluid and serum findings from forty-one patients in the acute phase of measles encephalitis and encephalomyelitis have been tabulated and discussed. Noteworthy is the fact that abnormal inorganic phosphorus values were revealed both in the cerebrospinal fluids and sera of these patients. A greatly increased level of inorganic phosphorus in the cerebrospinal fluid was found in eight out of twelve patients while the serum showed hypophosphatemia in nine out of eleven instances resulting in an abnormal ratio of cerebrospinal fluid inorganic phosphorus to serum inorganic phosphorus. The cerebrospinal fluid inorganic phosphorus values ranged from 1.7 to 4.1 mg. per cent. The mean was 2.02 mg. per cent with a standard error of the mean of ±0.11. When compared to a series of control patients previously reported this increase was found to be statistically significant. In addition, abnormally high cerebrospinal fluid total cholesterol values were found in three patients in the acute phase of the infection. The pathologic aspects of these findings and their application to therapy in the acute stage of the disease in these cases were discussed. The role

of magnesium in viral infections with central nervous system complications and its use in therapy was assessed. Also an unusually high percentage, 94 per cent, of the cerebrospinal fluids from our cases of measles encephalitis and encephalomyelitis revealed pleocytosis, elevated total protein, or both.

The characteristic white cell count in measles encephalitis and encephalomyelitis is that of an acute infection with an elevation of the white cell count with a tendency toward a relative granulocytosis. Where the white cell count remains normal, a relative lymphocytosis predominates. The serum constituents, glucose, total protein, albumin-globulin ratio, chlorides, urea nitrogen, sodium, potassium, calcium, and cholesterol, were, except for a few instances, within normal limits during the acute stage of the disease. Slight acidosis was found in most patients on admission.

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THE USE OF THE GARDNER-MURPHY PEDIATRIC SCALP-VEIN INFUSION OUTFIT

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INTRAVENOUS infusion in infants and young children oftentimes is a taxing technical problem in a pediatrician's everyday practice. Since parenteral fluid therapy to correct fluid and electrolytic imbalance has progressed to the extent that one can now adequately provide for a severely ill patient's daily metabolic and caloric needs, an improvement in the technique of administering intravenous therapy to infants and young children is a welcome boon to the busy pediatrician.

There are various methods and techniques of administering fluids by vein.[1] The scalp veins of infants are often the most accessible sites for intravenous infusions. The difficulty usually is in fixing the needle, adapter, and tubing in position securely enough to hold and stay functioning. This usually entails restraints, sandbags, complete immobilization of the patient, and constant nursing care. In spite of the best of care scalp vein infusions in this manner do not stay in the vein very long.

In 1951, Gardner and Murphy[2] devised a scalp-vein needle unit especially adapted for pediatric use. A review of the pediatric literature of the last two years has failed to show any published report of the use of the Gardner-Murphy Pediatric Scalp-Vein Infusion Outfit. This lack, therefore, has prompted publication of the paper.

Description.—The Gardner-Murphy unit consists of a polyethylene tubing about 12 inches long (diameter of lumen—0.034 inch) to which is attached on one end a short-beveled needle (gauge 22 or 23) without the usual hub. On the other end of the tubing is a modified metal adapter which can be attached to the glass adapter of a conventional intravenous infusion set. (See Fig. 1.)

We have used a modified version of this unit made manually by filing down the hub of different sizes of short-beveled 1 inch needles until they are just a little larger in diameter than the lumen of the polyethylene tubing and will fit snugly when inserted into one end of the tubing. On the other end a conventional 2 inch needle (gauge 18 or 19) cut to about 1 inch to eliminate the sharp edge is attached. (See Fig. 2.)

Sterilization.—The polyethylene tubing is the same type as that used in replacement transfusions,[3] as cannulas in surgical cut-downs, and other procedures,[1] and as stomach tubes in tube feeding of premature infants.[4] As it is plastic in nature, it does not stand boiling or autoclaving. The whole unit is therefore rendered aseptic by rinsing with sterile saline, and a 1:1,000 solution of Zephiran is drawn into the lumen of the tubing. The whole unit is then immersed in this solution in a covered receptacle for at least twenty-four hours before use. Before its use the unit is taken aseptically from the receptacle and washed externally and internally with sterile saline solution.

Fig. 1.

Fig. 2.

Fig. 3.

Technique of Application.—The area over the scalp vein is shaved and prepared aseptically. The infusion unit (previously washed with sterile saline solution after being taken from the receptacle) is attached to a 5 or 10 c.c. syringe filled with sterile saline solution. A few cubic centimeters of the saline is then forced out of the unit to check the patency of the needle.

Grasping the needle, preferably with a small snap forceps, the scalp vein is piereed. Due to the venous pressure the blood will stream out into the tubing the instant the needle enters the vein, and one can instantly see when he has successfully entered the vessel. A few cubic centimeters of the saline solution is then slowly introduced to make sure that the needle is in place before proceeding to tape it flat against the infant's head. Because of the light weight of the needle and the tubing, taping is relatively easy and is more securely done. The syringe is then detached and the glass adapter end of the infusion set is connected. (See Fig. 3.)

COMMENTS

During the last year and a half we have done more than a hundred intravenous infusions using this polyethylene scalp-vein needle unit. Using the sterile precautions described we have not observed any local tissue reaction or generalized infection attributable to the use of the unit. Intravenous infusions have been made with relative ease since adopting this devise, where previously cut-downs were frequently necessary. A cut-down is now rarely necessary.

Another advantage of this method is the relative comfort to the patient. The head can be left mobile without danger of the needle coming out if sufficient slack is allowed for the polyethylene tubing to absorb the movements of the infant's head (Fig. 3). The scalp-vein needle has been left in the scalp for as long as three to five days without any untoward effects noted.

SUMMARY

A report on the use of the Pediatric Scalp-Vein Needle Unit devised by Gardner and Murphy is presented. Satisfactory clinical experience with a slightly modified form has prompted this report.

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GENERALIZED CYTOMEGALIC INCLUSION DISEASE

Report of a Case and Review of Literature

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INTEREST in the incidence, extent, and clinical features of cytomegalic inclusion disease has increased in the last few years. Along with this has been a gradual evolution of views and concepts of its characteristics as a "clinical entity."

Inclusion bodies in the organs of many infants were first described as a protozoan-like cell by Jesionek and Kiolemenoglou in 1904.[1] The finding of these cellular inclusion bodies at autopsy in the salivary glands of infants[2, 3, 4] has added the phrase *"of salivary glands"* to its description. However, 1 to 2 per cent of unselected cases showed the presence of these bodies in other organs.[2, 3] The similarity of these inclusion bodies to the intranuclear cells of varicella, described in 1906 by Tyzzer[5] and in 1920 by Jackson[6] as a "protozoa," and still later in 1926 by Cole and Kuttner,[7] their isolation as filtrable viruses from the submaxillary glands of guinea pigs, led to a multiplicity of names: *"inclusion disease," "generalized salivary gland virus disease," "salivary gland virus infection," "protozoan cell-like disease,"* etc., and often with other modifying phrases as: "of infancy," "in adults," etc.

Reports in recent medical literature have lifted off some modifying terminologies from their original pertinence. Adults have been more and more reported to be victims of this disease.[8, 9, 12, 17] Since Goodpasture and Talbot[10] used the term "cytomegalia" in 1921 to describe a peculiar microscopic finding of cytomorphosis, a recent move has been initiated by Wyatt and associates[11] to settle this confusion and plethora of names by using a more descriptive term, "cytomegalic inclusion disease," with qualifications as to its being localized, generalized, fatal, or symptomatic.

Table I. Incidence of Organ Distribution

Organs involved with cytomegalia	Worth and Howard	Smith and Velios	Kidder	Wyatt and associates	This case
Kidneys	14	30	22	27	Yes
Liver	15	25	21	22	Yes
Lungs	15	51	18	53	Yes
Pancreas	8	12	12	10	Yes
Thyroid	6	10	10	7	Not done
Salivary gland	2	6	3	3	Not done
Adrenal	2	5	3	7	Yes
Brain	2	4	2	3	Not done
Pituitary	1	1	2	–	Not done
Epididymis	2	2	1	1	Not done
Ovary	–	–	1	–	Not done
Gastrointestinal tract	2	3	1	2	Not done
Skin	–	1	–	–	Not done
Spleen	2	2	–	–	Yes
Sweat glands	1	–	–	–	Not done
Bone marrow	1	–	–	–	Not done
Heart	1	–	–	–	Yes
Thymus	–	–	–	–	Yes

While the generalized form involves many organs as shown in Table I, it has been least frequently found in the heart and thymus. The present case has become of interest because it is the first case reported with thymic involve-

ment, and the second in record with cardiac inclusion body localization. Further, the picture of adrenal insufficiency in its course is an interesting feature.

CASE REPORT

K. S., 10 weeks of age, was admitted on Feb. 1, 1952, to St. Anthony's Hospital, for convulsions and cyanosis. The illness began with a cold a week previous and this was followed with difficulty of breathing on the night before admission. Early in the day she became cyanotic and developed convulsions.

When 4 weeks old, a dry skin was noted which slowly became reddened and thickened, and later some scaling and oozing were noted. Elixir pyribenzamine and zinc oxide ointment were prescribed. When 8 weeks old, the infant was not improving, and a change was made to Liquid SMA instead of Mull-Soy but was later changed back because the skin lesions became worse. Pyribenzamine ointment was used on the skin.

Physical examination showed some suggestion of rigidity of the neck and spine. Hyperemic and desquamative skin lesions were present all over the body. Loud coarse rhonchi were heard all over the chest but no definite râles were present. A loud systolic apical murmur was audible. The liver was palpable 2 fingerbreadths below the right costal arch. Laboratory data on that day were: red blood count, 4,680,000; white blood count, 18,000, differential count, 41 stabs, 27 segmentals, 32 lymphocytes. The spinal fluid was clear, containing one cell per cubic millimeter. Therapy consisted of Campenamine (penicillin), Mull-Soy, and an ointment containing lanoline, starch, boric acid, and talc; nebulized oxygen inhalation and elixir pyribenzamine.

The following day the stools became foul smelling and frequent. A chest plate was negative. The skin lesions became scaly and dry fissures developed over the popliteal areas, and the cheeks had a scaly erythematous blush. Neck rigidity was definite. Carbon dioxide combining power was 23 vol. per cent, and 1/6 molar sodium lactate and glucose with Alidase was given. The dermatitis and respirations improved, but the loose movements increased. Spinal fluid on February 12 showed a clear fluid with 2 cells, sugar 64 mg. per cent, proteins 125 mg. per cent, and chlorides 688. Chloromycetin was given to replace penicillin. The neck rigidity, fever, and bowels were worse. With Campenamine resumed, aspirin and sodium molar lactate being continued, and urine smear showing gram-positive diplococci, Gantrisin was added to the therapy.

On March 2, the urine still showed gram-positive diplococci and gram-negative rods. The red blood count was 2,060,000 and the white blood count 7,000 with 21 stabs, 33 segmentals, 43 lymphocytes, and 3 eosinophils. The stools became more explosive and dehydration worse. Dihydrostreptomycin was started. The diet was changed from Mull-Soy to Olac. Blood plasma was given. The fever remained very high, and the liver and spleen became more palpable. On March 16, the temperature was down; antibiotics were stopped but had to be resumed on March 18 when temperature went up to 104° F. Plasma, glucose in Ringer lactate, Gantrisin, and Terramycin were prescribed. A blood transfusion was given and Vioform ointment applied to the skin. The general condition improved.

On this regime there was little change for two weeks. In early April the red blood count was 4,040,000; white blood count 4,500 with 12 stabs, 24 segmentals, 60 lymphocytes, and 4 monocytes. The carbon dioxide combining power was 31 per cent and the blood showed sodium 315 mg. per cent, potassium 27.1 mg. per cent, and chlorides 712.5 mg. per cent, suggesting a picture of hypoadrenalism. DOCA was started along with a celiac routine, after obtaining a positive film test of the stool. Casec, Olac, banana powder, thyroid, testosterone,

Fig. 1.

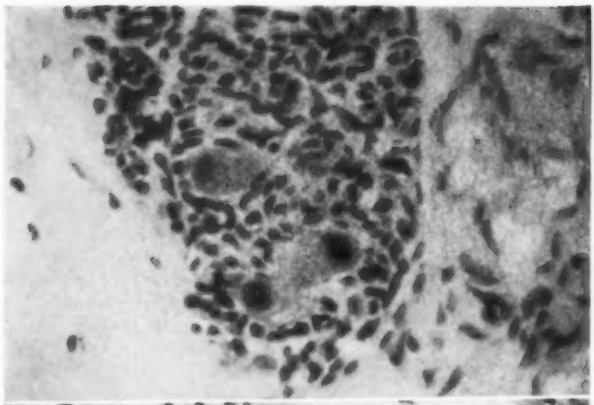

Fig. 2.

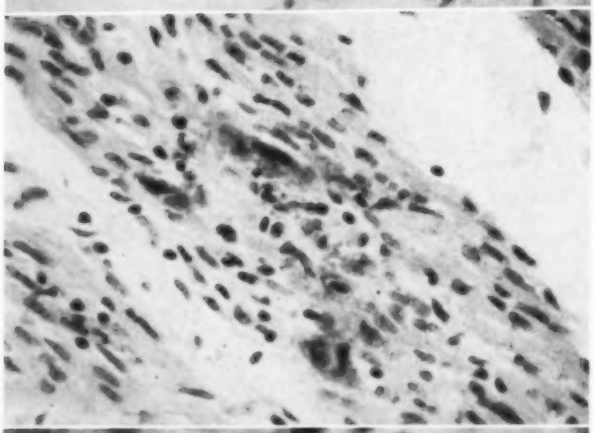

Fig. 3.

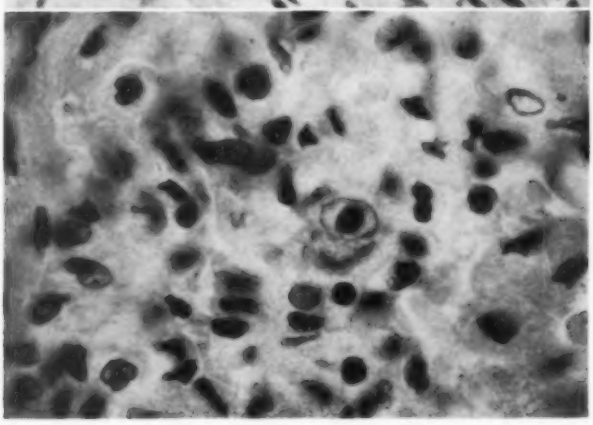

Fig. 1.—Thymus showing three large cells with discrete cytoplasmic masses.
Fig. 2.—Inflammatory lesion of myocardium.
Fig. 3.—Higher power to show the cellular detail in myocardium.

and vitamins were started. A blood culture was negative. The patient worsened rapidly and died on April 20, 1952.

Fig. 4.

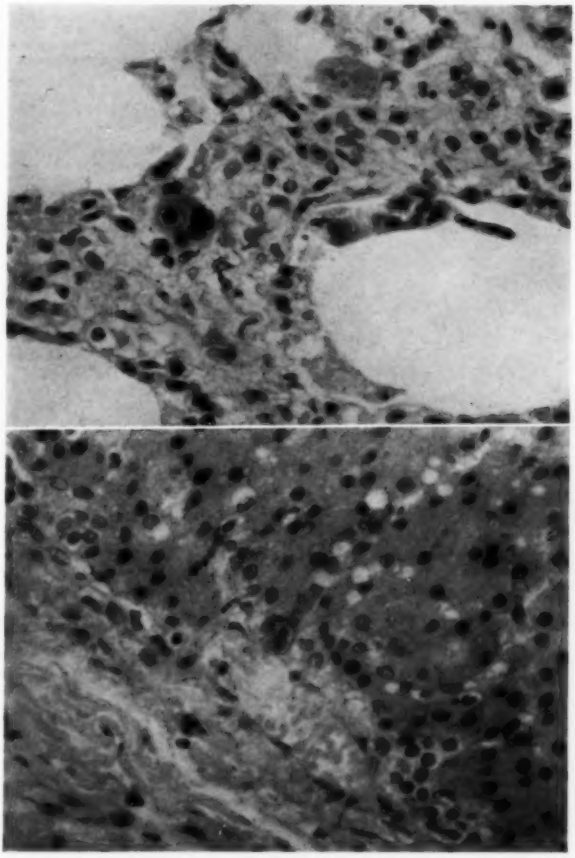

Fig. 5.

Fig. 4.—Interstitial pneumonia with inclusion-bearing cells of the lung.
Fig. 5.—Individual cell in liver.

Post Mortem (B. C. Portuondo, M.D., Pathologist). — Pertinent autopsy findings on gross examination were limited to extreme emaciation, as shown by marked prominence of all bony landmarks. There was a moderate degree of desquamation of the skin of the chest. Internal examination revealed a very marked atrophy of the thymus which was difficult to evaluate grossly. The heart weighed 20 grams, and the musculature of the left ventricle was somewhat softer and somewhat more yellowish than is ordinarily encountered. The lungs showed nothing of significance and the liver, which weighed 110 grams, was of slightly more yellowish color than usual.

Microscopic examination of the tissues showed essentially two lesions. There was granulomatous reaction in

one, and free inclusion-bearing cells in the other. In the first category, the *heart* muscle showed areas of basophilic staining necrosis infiltrated with owl-eyed cells, lymphocytes, monocytes, and very occasionally with neutrophils. cross striations could be followed around the inclusion-bearing cell but not through it. The *pancreas* showed one small focus quite similar to that found in the heart although there was less leukocytic infiltration in

Fig. 6.

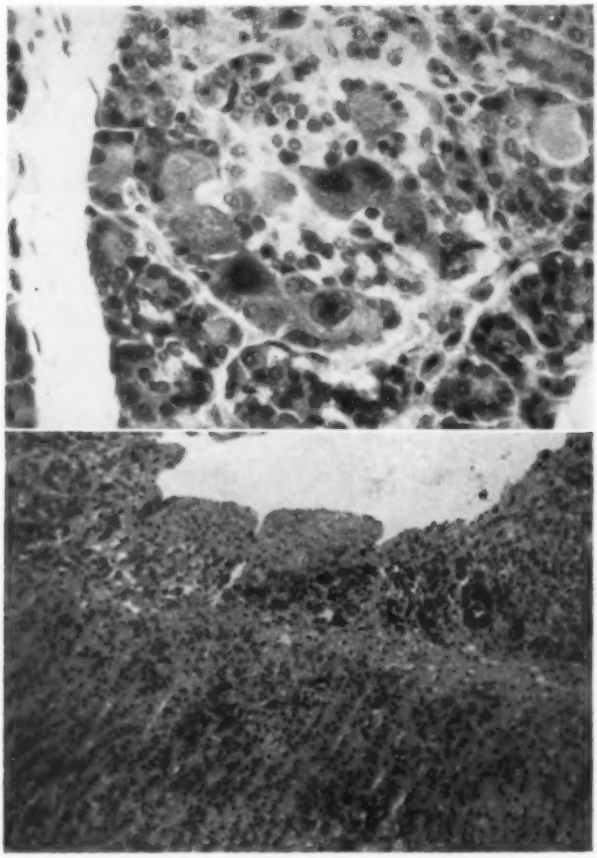

Fig. 7.

Fig. 6.—Pancreas showing cellular infiltration.
Fig. 7.—Appearance of adrenal with infiltration.

In addition to this granulomatous reaction, there were several of the large inclusion-bearing basophilic staining cells found either between the myocardial fibers or actually separating the cytoplasm of the cell so that the the pancreas. The other organs showed the inclusion-bearing cells lying by themselves. Sections of the *thymus* showed small lymphoid follicles with considerable fibrosis and collections of two or three of the cyto-

megalic cells without surrounding reaction. The *splenic follicles* were small and only occasional inclusion-bearing cells were found. The *liver* sections showed only two of the large basophilic staining cells. Sections of the *lungs* showed an interstitial pneumonia with giant cells of two types: the one, being larger and containing vesicular large nuclei around the periphery; and the other, being much smaller and containing small basophilic staining nuclei tending to be central in position. Individual large inclusion-bearing cells were found in the alveolar air sacs as well as in the walls of the alveoli. A section of one of the tracheobronchial *lymph nodes* showed one large inclusion-bearing cell. Sections of the *adrenal* showed a concentration of a fairly large number of inclusion-bearing giant cells in the medulla of either side, with some necrosis of the medullary structure. In the *kidneys*, one glomerulus was found containing an inclusion cell together with several within the lumina of the convoluted tubules and collecting tubules.

DISCUSSION

The clinical picture of our case is as protean as it is bizarre in its inclusion body distribution. Like most of the reported cases of generalized form, the systemic involvement, *a posteriori*, reveals the possibility of symptomatic causation due to their presence, to wit: dermatologic findings, gastrointestinal disturbances, cerebral symptoms, respiratory difficulties, renal and adrenal involvement, etc., when these cytomegalic inclusion-bearing cells were found in the skin, alimentary tract, brain, lungs, kidneys and adrenals, etc., respectively. Thus we can look back and note that the chronic eczema of our patient since the age of one month, the persisting diarrhea, the respiratory element in the picture, the cerebral signs, the renal and adrenal components, etc., unabated in spite of treatment, may well be explained by the involvement in each system. In like manner can we agree to the implied claims of the respiratory form as reported by Gallager,[18] Wyatt and coworkers,[12] and Smith and Velios.[19]

A study of the organ distribution of this disease shows that it is more generalized than localized in the salivary glands and other organs. Table I shows the predominance of their findings in the kidneys, lungs, liver, etc., as summarized from the four extensive reviews of Wyatt and associates,[11] Worth and Howard,[14] Kidder,[15] and Smith and Velios.[19]

Against earlier claims[20] that no clinical effect had been produced by the agent responsible for the nuclear changes, it is now safe to say that local signs and symptoms, either directly due to viruses or as a reaction of tissues to the cytomegalic inclusion-bearing elements, were present in most cases. Whether or not related to the cause of death, they were in all probability related to the clinical course and picture of disease. Thus, if we classify the symptomatic presentations of cases according to systems involved, we may justifiably create clinical forms of the disease depending upon the predominant system chiefly affected by these viruses or by the presence of these abnormal owl-eyed giant cells.

The *respiratory form* has been presented in a variety of pictures, as pertussis, pneumonia, diphtheria, lung abscesses, etc. Dyspnea, cyanosis, x-ray findings, râles, and mucoid or bloody expectorations have been the usual findings. Gallager[18] reported the first respiratory form ever cured by surgery —a case of pulmonary cysts ending in the removal of the lung which on microscopy was found to contain cytomegalic inclusion-bearing elements.

The *cerebral type*, as observable in the reports of Kinney,[13] Worth and Howard,[14] Wyatt and associates,[11] Kidder,[15] and Fetterman,[16] presents in a variety of central signs and symptoms evidencing hydrocephalus, cerebral hemorrhage, convulsions, etc.

The *gastrointestinal form* has been presented in the persisting incidence of diarrhea, colitis, pancreatic fibrosis, hypoplasia of the ileum and colon, ulcerative lesions of the intestines, etc.

The *dermatologic type* has been shown in the forms of chronic eczema, purpura, petechiae, ecchymosis, scaling, gangrene, etc. The conjunctiva has also been found to have xerophthalmia or conjunctivitis.

The *renal* involvement has been in the forms of nephritis, infiltration of the kidneys with these cells, hematuria, etc. Fetterman[16] is the first to have diagnosed this disease by urine examination and smear. If we refer to Table I, we note that many of these cases have renal involvement. Perhaps routine smears, as Fetterman has advocated, may pick up the diagnosis, especially in therapy-resistant cases of no known rationale.

The *adrenal form*, although rare, may be occasionally encountered, as is this case. The picture of adrenal insufficiency may be explained, on hindsight, by the presence of these inclusion-bearing cytomegalia which in other cases may have caused the focal necrosis of the adrenals.

The *hepatobiliary form* is one of the most common clinical findings. Jaundice, hepatic fibrosis, cirrhosis, necrosis, cholelithiasis, and the frequent association with splenomegaly cannot escape the attention of one reviewing reported cases in the literature.

The *hematologic* participation of this entity has been in the forms of multiple hemorrhages, purpuras, ecchymosis, anemia, hemolytic diseases, leukemia, erythroblastosis, etc.

The contributory findings of thymic involvement and cardiac participation may have added to the severity of the case and the poor prognosis.

Pathology.—This disease is still a purely pathologic entity, its diagnosis being mainly established by a cytomegalia "too impossible to overlook in a microscopic examination."[20] Both the cell body and nucleus are gigantically enlarged as contrasted with the surrounding structures. The inclusions are large acidophilic or basophilic masses centrally intranuclear in location, with their enlarged nuclei haloed by a clear zone of vacuolated or granulated cytoplasm which is peripherally ringed by chromatinic massing along the nuclear membrane, thus presenting an aspect of an owl's eye.

An observation in the present case is the presence of two varieties of lesions: (1) granulomatous in nature, as exemplified in the myocardium and suggested slightly in the pancreas; and (2) infiltration of other organs by large inclusion-bearing cells without any reaction at all. While this could be explained on the basis of age of the lesion, there is also a possibility of the pathogenicity of the invading agent or of the tissue reaction to its invasion. (See Figs. 1 to 7.)

Etiology.—The etiology of this disease is considered viral in nature. The histopathology of other intranuclear inclusion-body-bearing diseases as in inclusion blenorrhea, chicken pox, and occasional virus pneumonia is similarly comparable with those found in this disease.[20] However, in human beings, no experimental transmission nor virus demonstration by inoculation of embryonated eggs or tissue culture has been successful.[19]

Diagnosis.—Diagnosis is based purely on the histopathology. It has been suggested that any cases suspected to be of this kind should be given adequate virus study. Only urine studies and smears, as done by Fetterman,[16] has, up to this time, been a laboratory aid of proved validity.

Treatment.—Treatment of the disease is difficult as the cause and localization cannot well be determined. However, surgery has succeeded, as reported by Gallager.[18] In the meantime, no known specific therapy is available. Symptomatic treatment is the rule.[21]

Terminology.—As first advocated by Wyatt and co-workers,[11] "cytomegalic inclusion disease" very well projects the histopathologic picture into the minds of clinicians. In view of the prevalence of these elements in organs other than the salivary glands, the old portion *"of salivary glands"* in the original terminology should be dropped off. The increasing incidence of adult involvement[8, 9, 12, 17] nullifies

the *"of infancy"* pertinence to its first name. For brevity and inclusiveness of meaning, this disease should be called "cytomegalic inclusion disease" whether local or generalized, symptomatic or fatal, of adults or of infants.

COMMENT

This diagnosis was not suspected, hence no virus studies were made before or soon after death. The persisting picture of diarrhea not abated by therapy, the resultant dehydration, the creeping picture of adrenal insufficiency not improved by DOCA, the chronic dermatologic lesion not improved by antiallergic and other ointments, all made this case a clinical puzzle.

The histopathologic finding of owl-eyed giant cells with intranuclear inclusion bodies in the different systems involved could explain, looking backward, almost all of the complex symptomatology. The illustrations shown are identical with those reported in previous literature. Prof. Pinkerton and his staff, in the Department of Pathology of the St. Louis University School of Medicine, have reviewed and confirmed our slides. It is to be regretted that no specimens from the skin, brain, bone marrow, and sweat and salivary glands were obtained.

SUMMARY

A case is reported of protean manifestation diagnosed at autopsy as a generalized form of cytomegalic inclusion disease. It is the second with cardiac involvement and the first with thymic localization.

A summarization of reported cases, with the view of classifying their clinical pictures according to systems first or mainly involved, has been done.

A brief recapitulation of the main pathologic finding in this entity, with a comment on the etiology, diagnosis, and treatment, has been presented.

Gratitude is hereby expressed to Dr. B. C. Portuondo, Pathologist of St. Anthony's Hospital, St. Louis, Mo., for advisement in the preparation of this paper; to Prof. Pinkerton and his staff, in the Department of Pathology, St. Louis University Medical School, for their confirmation of our slides and for the photomicrographs used in this report; and to the Medical Record Department, St. Anthony's Hospital, St. Louis, Mo., for facilities rendered in this case record.

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TREATMENT OF STAPHYLOCOCCIC PERICARDITIS WITH BACITRACIN

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THE purpose of this paper is to present a case of acute staphylococcic pericarditis with pericardial effusion which was treated successfully with bacitracin following the unsuccessful use of large doses of penicillin and aureomycin. Friedberg and Bader[1] have reported the results of a case of staphylococcic pericarditis which was cured by bacitracin used in conjunction with large doses of penicillin. An increasing number of reports[2] indicate that more and more strains of staphylococcus are becoming resistant to penicillin, heretofore the preferred antibiotic against this organism. Reports both here[3, 4] and abroad[5] indicate that there has been a striking increase in the percentage of strains of penicillin-resistant staphylococci. In a review of the literature on the use of bacitracin, we were unable to find any reports concerning the systemic use of bacitracin alone as therapy for this condition. In all reported cases bacitracin was used in conjunction with other antibiotics.

CASE REPORT

R. C., a 7-year-old white male child, was seen on July 7, 1952, with the chief complaints of "not feeling well" and vomiting after play. He was given an enema at this time by his stepmother. She noted that the child had respiratory difficulty while sitting up and was unable to lie down. However, when the child fell asleep shortly thereafter these findings disappeared. That evening the child again began to have respiratory difficulty and was seen in the outpatient department of a local hospital. The physical examination at this time was completely negative. A blood count revealed a moderate anemia. The child was sent home for observation on a small amount of aspirin. On the day of admission, July 8, 1952, he was seen in the office by one of us. The stepmother stated that the respiratory difficulty had persisted throughout the night, and it was noted that he was better able to breathe sitting up than lying down.

On physical examination at the office his respirations were of a peculiar gasping nature with dilatation of the ala nasi, intercostal retraction, and diaphragmatic breathing. The physical examination was otherwise negative except for questionable tenderness to deep palpation in the left upper quadrant.

Past History.—The child was a full-term, normal delivery, one of a set of identical twins. Development had been satisfactory throughout his life, though his twin seemed to be somewhat more advanced than his brother. The mother had died of multiple sclerosis approximately one year before and the children were placed under the care of a housekeeper. There were the usual childhood diseases. The twin brother was admitted to the hospital in the spring of 1952 and a diagnosis of Sydenham's chorea was made. The father was living and well.

Physical Examination.—The physical examination three hours after admission to the hospital was negative except for a heart rate of about 90 and a questionable tenderness to deep palpation in the left upper quadrant.

Laboratory and X-ray.—The admission x-ray of the chest showed a normal heart shadow and diaphragm. There was a small dense area to the

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right of the mediastinum at the level of the arch of the aorta. The lung fields were otherwise clear. Fluoroscopy at this time revealed that the mass did not fluctuate and did not appear to be attached to the heart. The electrocardiogram on the day of admission revealed a slight elevation of the entire RST segment in SL_1, SL_2, aV_F, and V_5 and the heart rate was 125. The P-R interval was 0.14 second. The conclusion was sinus tachycardia with possible early pericarditis. A blood culture was also done on admission. The urine report was negative. Hemogram revealed 10.6 grams of hemoglobin, 4.31 million red cells, 10,150 white cells with 86 per cent polymorphonuclear leukocytes and 14 per cent lymphocytes. The hematocrit was 38 and the corrected sedimentation rate was 34 mm. per hour.

The temperature on admission was 101.8° F. and ran a septic course with peaks to 104° daily. The pulse fluctuated between 100 and 160 beats per minute. The child was placed on aqueous procaine penicillin, the dosage of 300,000 units every twelve hours.

On July 11 examination of the chest by x-ray revealed early enlargement of the heart shadow, and the electrocardiogram showed no fundamental changes from that previously reported.

The same day when the admission blood culture was reported as positive for *Staphylococcus albus,* coagulase negative there was a question of it being a contaminant and it was decided to withdraw four more blood cultures in the succeeding twenty-four hours and to discontinue penicillin while this was done.

On July 12 the heart sounds were muffled and the clinical condition was much worse. Because of the gravity of the clinical picture the child was started on 300,000 units of aqueous crystalline penicillin every three hours. In spite of this therapy he continued to spike a daily temperature to 104° F. On July 13 a friction rub was present for the first time along the left sternal border at the fifth rib. Respirations at this time were labored and of poor quality and oxygen was started. The heart rate was 160 beats per minute. On July 15 aureomycin was started by the intravenous route in 100 mg. doses as well as 250 mg. of oral aureomycin daily. In spite of the therapy with penicillin and aureomycin in what was considered to be adequate amounts, the child's condition was progressively downhill.

By this time two of the blood cultures of July 12 were reported back positive for hemolytic *Staph. albus,* coagulase negative, and when run for sensitivity it was found that the organism was completely insensitive to all known chemotherapeutic agents except bacitracin. In view of this, bacitracin for intramuscular injection was obtained. This was started in the recommended dosage of 400 mg. per kilogram of body weight. Penicillin and aureomycin were discontinued. The child received 6,000 units of bacitracin every six hours intramuscularly. Because of the reported nephrotoxic effects[6] a urinalysis was done every twenty-four hours and a nonprotein nitrogen test every forty-eight hours.

By July 20 the clinical picture had not improved. Two new developments then occurred. The child began to complain of pain under the sternum, most marked on the left side, and another and louder friction rub could be heard over the apex of the heart. The parasternal friction rub at the fifth rib was still present.

An x-ray on July 20 revealed marked enlargement of the heart shadow, especially on the left side. This was interpreted as suggestive of pericarditis with effusion. The lung fields were clear. An electrocardiogram on July 21 remained unchanged.

On July 20, because of the ineffectiveness of the bacitracin at that time, it was felt that the dosage

should be increased in spite of the nephrotoxicity. Ten thousand units every six hours was given.

On July 21 the temperature spiked to 102° F., and on the following day the child was clinically improved. The precordial pain diminished in spite of the persistent friction rubs. From this time on the child gradually improved. The temperature was normal for the first time on July 26. It was noted at this time that there was a gradual diminution in the friction rub over the apex and the dullness in the left chest was less marked. The heart sounds became stronger and the rhythm and rate more firm and regular.

An x-ray on July 29 revealed that there was some decrease in the size of the heart shadow since the previous examination. However, it was still definitely enlarged with a distinct left margin of the heart and well areated left lung field.

On August 1 the friction rub had disappeared completely. Five days later whispered pectoriloquy was heard over the left chest posteriorly. There were also absence of breath sounds and dullness to percussion over the left chest posteriorly. An E to A change was also present. On August 6 x-ray examination showed a slight decrease in the size of the heart shadow, but there was increased density in the left lower lobe.

The heart rate was 96 and the respirations were 36 at this time. It was felt that the child had a compression atelectasis or pneumonitis from the previous massive enlargement of the heart and pericardial sac.

On August 13 the x-ray was read as within normal limits for the heart shadow, and the right lung was well areated. However, the left lung field revealed some peribronchial increase at the level of the lower pole of the hilum on the left side. At this site there was a questionable atelectasis or pneumonitis as on previous films.

Since the original need for bacitracin now seemed to be ended, e.g., normal heart size and findings and normal blood cultures, it was felt that it should be discontinued and at the time we decided to give him dihydrostreptomycin prophylactically, 150 mg. every six hours for the questionable pneumonitis that had developed.

On August 21 an x-ray of the chest revealed a normal heart shadow and well-areated lung fields. An electrocardiogram taken on the same day was read as being within normal limits.

On August 17 300,000 units of aqueous procaine penicillin was started for prophylaxis. This was later changed to 300,000 units of oral penicillin twice daily at the time of discharge for home use.

Temperatures remained normal for the remainder of the hospital stay. Seven more blood cultures were reported as negative and an x-ray on September 3 was also negative.

Nine months following discharge from the hospital the child has continued to gain weight, is active, and has had no complaints. Physical examination has remained negative except for a systolic murmur over the mitral area.

SUMMARY

This is a case of hemolytic *Staphylococcus albus*, coagulase negative, bacterial pericarditis with pericardial effusion treated successfully with bacitracin alone. Although the drug was used in doses larger than that recommended by the company, no apparent permanent ill effects occurred in this case. We feel that this drug will be used more often as the resistance of organisms to other antibiotics becomes more pronounced.

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PRIMARY CARDIAC TUMORS IN INFANCY

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PRIMARY tumors of the heart in infants are very rare as is attested to by the paucity of case reports on this subject in the literature. Nevertheless, their existence, even though in small numbers, warrants their consideration in the differential diagnoses of cardiac enlargement or cardiac failure during infancy, if for no other reason than that the scope of cardiac surgery has broadened sufficiently in recent years to make feasible early surgical extirpation of such lesions with the hope of achieving an occasional cure.

In the past year a case of rhabdomyosarcoma of the right ventricle was encountered at the Children's Hospital in a 3-month-old infant. The most common types of primary cardiac tumors which may be found during early life, and the diagnosis and management of infants with such lesions form the basis of this report.

TYPES OF PRIMARY CARDIAC TUMORS

Because of the relative rarity of primary cardiac tumors in every age group, there is insufficient data available in most instances, except for the comprehensive studies of Pritchard[1] and Mahaim[2] from which to draw any conclusions as to which type of cardiac tumor is most likely to be encountered during early life or in the younger age groups. Nevertheless, a review of the more common primary cardiac tumors may prove helpful in defining the nature of these lesions.

Rhabdomyoma.—Of all the primary cardiac tumors that have been described, rhabdomyoma appears to be the only one which shows a definite predilection for the younger age groups; particularly is this true in cases of children suffering with tuberous sclerosis in whom rhabdomyoma of the heart is most prone to occur.[3] Such tumors are not considered true neoplasms, but instead probably represent an area of developmental arrest in the fetal myocardium. It is not unusual for rhabdomyoma to regress spontaneously without causing any appreciable impairment of cardiac function.

Myxoma.—By far the most frequent primary tumor of the heart is the myxoma which accounts for slightly more than half of all primary cardiac tumors. They may be encountered at almost any age from early infancy to late adult life.

The signs and symptoms of these tumors vary rather widely, depending on the age of the patient, but ultimately they generally lead to cardiac failure which is irreversible and does not respond to digitalis or any other form of medical therapy.

The vast majority of such myxomas are located in the atria, more frequently in the left atrium. They tend to proliferate and project into the chambers of the heart, thus preventing normal cardiac filling, and on occasion to obstruct the mitral or tricuspid valve orifices.

The origin of these tumors is still an extremely controversial subject but on the basis of histologic studies which have revealed that the bulk of cardiac connective tissue is to be found concentrated in the auricular septum it has been postulated by Pritchard[1] that the auricular septum would seem to be the most logical site of origin for such cardiac connective tissue tumors as the myxoma.

From the Surgical Service of the Children's Hospital, and the Department of Surgery, Harvard Medical School.

Sarcoma.—Primary sarcomas of the heart are slightly less common than myxomas but like cardiac myxomas they occur at all ages from infancy to late adult life.

The signs and symptoms produced by these tumors are directly related to their location, which most commonly is in the right auricle or right ventricle. Unlike myxoma they do not, as a rule, proliferate in an intracardiac direction, but instead infiltrate the wall of the myocardium and extend outward into the pericardial cavity frequently involving the pericardium.

Though primary sarcomas of the heart arise from mesenchymal tissue, not infrequently gross and microscopic examination of these tumors reveal a wide variation in their histologic character. Some appear to be composed predominantly of fibrous, myxomatous, or fibromyxomatous tissue, while certain others are composed chiefly of round cells resembling lymphosarcoma, and, still others may more nearly suggest the picture of a rhabdomyosarcoma.

Angioma, Fibroma, Lipoma, and Hamartoma.—These primary cardiac tumors are extremely rare but are included for the sake of completeness though their discussion is beyond the scope of this report.

CASE REPORT

T. B., No. 390418, a white male infant, was referred to the Children's Hospital from another hospital and admitted on Aug. 9, 1952, at the age of 3 months.

Family history was noncontributory, the patient being the third child born to a mother, 24, and a father, 25, both of whom were in good health. Two previous siblings, 2½ and 3½ years old, were in good health. The mother had had no previous miscarriages or stillbirths. The maternal great-grandmother was the only member of the family known to have died of cancer.

Past history revealed that this male infant was the product of an entirely normal and uneventful pregnancy and delivery. Birth weight was 5 pounds, 6¼ ounces. During the first two months of life the patient gained weight at the rate of 2½ to 3 pounds per month. However, during the four weeks preceding admission he gained only 2 to 3 ounces. Mental and motor development were normal during the first two months of life.

Present illness began after the second month of life when the infant began to vomit a significant proportion of all his feedings, failed to gain weight, developed anorexia, and refused any more than one or two ounces at each feeding. At this time the mother noted that the infant's cry was no longer loud and lusty but faint and weak. During the two weeks immediately prior to admission he had had repeated episodes of severe coughing, particularly after his feedings. During these coughing spells and when crying, the patient became markedly cyanotic, would stop breathing for a few moments, appear to be choking, and in imminent danger of dying. On Aug. 2, 1952, the infant was admitted to another hospital where the above symptoms persisted unabated. After x-ray films of the chest were taken, the possibility of an enlarged thymus was considered and the child was given 173 r through a 6 × 8 portal in air, with no improvement. Subsequently, cardiac fluoroscopy was carried out which suggested the possibility of: (1) a large tumor or cyst of the mediastinum or pericardium, (2) a greatly enlarged heart, (3) a large pericardial effusion, (4) duplication of the esophagus, (5) rhabdomyosarcoma of the heart, (6) endocardial sclerosis, or (7) glycogen storage disease. Six days later, the infant was referred to the Children's Hospital for further evaluation and treatment.

Physical examination revealed a very sick, male infant who was moderately cyanotic, and in obvious respiratory

distress. Temperature was 100° F., pulse 116, respirations 60. When the child was out of the oxygen tent, he became markedly cyanotic. Despite difficulties in feeding, the baby had been able to take some oral feedings, and, therefore, was only moderately dehydrated. There was no peripheral edema, but the neck veins were slightly distended. The child was breathing rapidly and was using his accessory respiratory musculature. There was dullness to percussion over the entire anterior chest and the breath sounds were absent except in the right mid-axillary thoracic region. Clinically, the heart was displaced or enlarged to the left. The heart sounds were not diminished over the left precordium. No murmurs were heard. The liver extended three fingerbreadths below the right costal margin. The spleen was not palpable. There were no ascites, nor was the abdomen distended. The remainder of the examination was essentially normal. X-ray examination and cardiac fluoroscopy were carried out the day of admission and this revealed that the esophagus and the trachea were displaced to the right, suggestive of a pericardial effusion (Fig. 1).

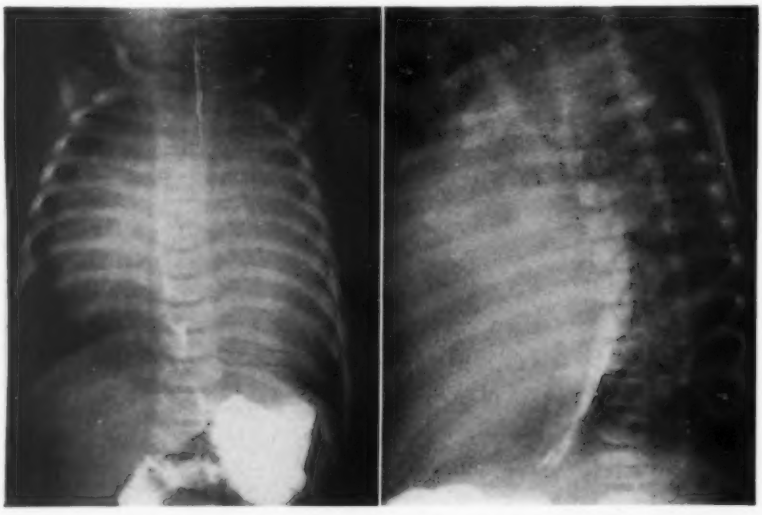


Fig. 1.—Anteroposterior and oblique films of chest at time of admission suggesting pericardial effusion with encroachment on both lung fields. The barium-filled esophagus is displaced posteriorly and to the right.

Since the infant's condition was deteriorating rapidly and his respiratory exchange was barely sufficient to maintain life, a left exploratory thoracotomy was carried out the same day. This revealed a compressed, partially atelectatic left lung and a tremendously distended pericardium. At this point the heart action slowed markedly, became extremely feeble, and the pericardium was incised rapidly in the hope of releasing the obvious cardiac tamponade. On opening the pericardium, a large mass of myxomatous viscid, grapelike clumps of tissue herniated through the defect (Fig. 2) and the heart action improved immediately. Without difficulty the remainder of the pericardial cavity was emptied of the myxomatous tissue, much of it being aspirated with a sucker. At this point it was possible to trace the pedicle of the tumor down to the upper border of

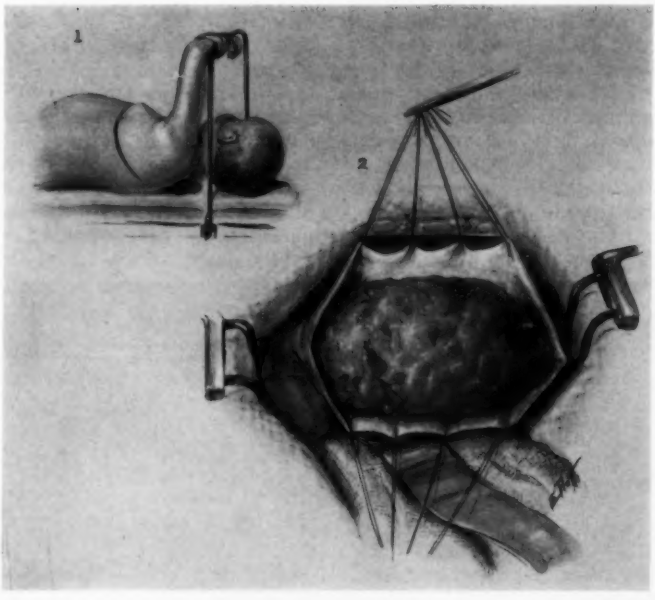


Fig. 2.—Operative approach and exposure of tumor protruding from the opened pericardial sac.

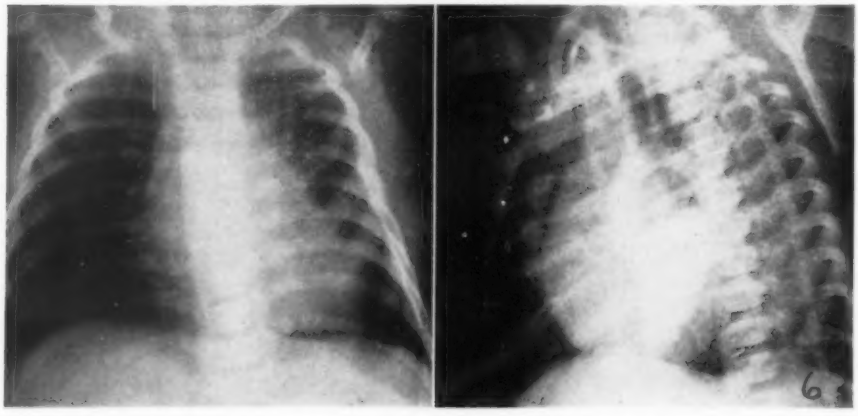
Fig. 3.—Photomicrograph of a section of the tumor removed at operation. (×125, reduced ⅙.)

the right ventricle where it joined the right atrium. The tumor pedicle was divided without incident but no further attempt was made to resect the portion of the tumor infiltrating the wall of the right ventricle, since frozen sections of the myxomatous tissue revealed it to be a highly malignant neoplasm (Fig. 3).

Postoperatively, the patient did extremely well, made an uneventful recovery, and had a marked diminution in his cardiac size with complete relief of his respiratory symptoms (Fig. 4).

Sixteen days after operation an angiocardiogram was carried out to determine if there was any intracardiac extension of the tumor, but no intracardiac filling defect was noted (Fig. 4).

During the latter part of the admission and following discharge, the patient was given a trial course of



A. *B.*

Fig. 4.—*A*, Roentgenogram of chest twelve days after removal of the tumor from the pericardial sac and wall of right ventricle. *B*, Angiocardiogram sixteen days postoperatively did not reveal a filling defect that would suggest intracardiac extension into either ventricle.

Fig. 5.—Roentgenogram six weeks after operation, revealing extensive recurrence of tumor.

antifolic acid therapy, specifically triethylene-phosphoramid (TEPA), since the malignant tumor had been incompletely removed, and it was not considered radiosensitive.

After discharge on Sept. 3, 1952, the patient did well for approximately one week, but thereafter he developed anorexia, began vomiting feedings, coughing again, and soon his respirations became very labored with frequent episodes of cyanosis. The infant was readmitted on Sept. 27, 1952. Physical examination on this second admission was rather similar to that of the first admission. As was expected, the roentgenologic examination revealed a rapid growth and extension of the sarcoma through

the mediastinum and adjacent left pleural cavity and lung (Fig. 5). The patient pursued a rapidly downhill course and died the following day.

Post-mortem examination of the heart revealed that the tumor had changed markedly in character and no longer consisted primarily of myxomatous tissue as was noted at operation, but instead on gross examination appeared to be composed almost entirely of fibrous tissue which had grown around and encapsulated the major portion of the heart. Though no intracardiac extension of the tumor was found, it did not remain localized within the pericardial cavity, but spread widely involving the left lung, the thoracic inlet, and the diaphragm rather extensively.

Microscopic examination of sections of the tumor tissue obtained at post mortem (Fig. 6) revealed small lobular collections of plump tumor cells that had a rather eccentric nuclei. The cytoplasm of these particular cells was stained a very intense eosinophilic hue. Frequently, the cytoplasm streamed in a broad band from one end of the cell and here and there an occasional mitotic figure was seen. The fibroblasts seen in these sections were rather immature, had an oval elongated nuclei, and a vesicular chromatic pattern with moderately large nucleoli.

In summary, the surgical specimen obtained seven weeks before death was diagnosed as a myxosarcoma, while the post-mortem tumor tissue described above more nearly resembled a fibrosarcoma with definite areas of rhabdomyosarcoma.

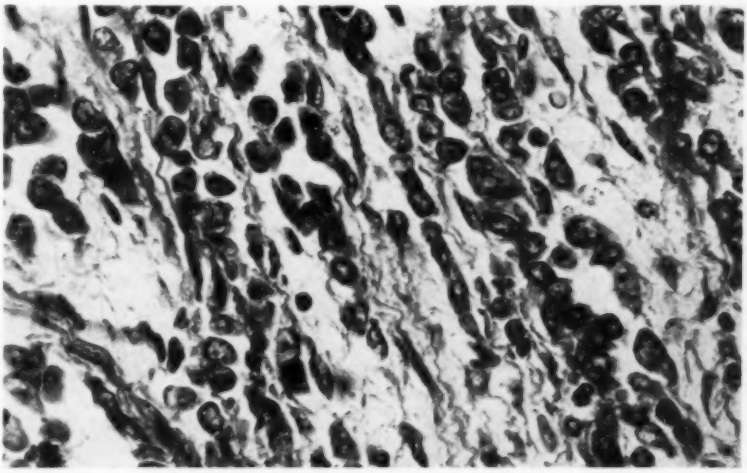


Fig. 6.—Photomicrograph of section of tumor tissue obtained from wall of right ventricle at post-mortem examination. (×425.)

DIFFERENTIAL DIAGNOSIS

As the preceding case report has shown, primary cardiac tumors in infants may produce a wide variety of signs and symptoms, depending on the number of organ systems affected. Indeed, only through early consideration of this diagnostic possibility and correct interpretation of these signs and symptoms will it be possible to salvage a significant proportion of these patients by prompt surgical intervention.

History.—In the presence of the aforementioned signs and symptoms, the history of normal neonatal and early life growth and development

may frequently provide the most significant clue available to suggest the possibility of a primary cardiac neoplasm. Though our experience is extremely limited, it appears that infants with such tumors often are considered entirely normal at birth with no evidence of cardiomegaly, tracheal or esophageal obstruction, or cyanosis. Subsequently, as the tumor proliferates in an intra- or extracardiac direction, signs of irritability, failure to gain weight, coughing, respiratory retraction, cyanosis, cardiomegaly, and ultimately cardiac failure intervene to alter the previously normal pattern of growth and development. In the face of such a history the possibility of a primary cardiac tumor should always be considered despite its statistical rarity.

Cardiomegaly.—Primary cardiac tumors may project into the chambers of the heart obstructing or impairing its function as a pump, or may proliferate peripherally, filling the pericardial cavity and ultimately causing tamponade as in this case. In either situation gradual cardiac enlargement occurs, cardiac filling is usually diminished, and congestive failure and death ensue. Such cardiomegaly considered alone may resemble in many respects or be indistinguishable from the infantile cardiac form of von Gierke's disease,[4] idiopathic myocarditis,[5] endocardial sclerosis,[6] or the generalized cardiac dilatation which occurs in certain types of severe congenital heart disease with or without failure.

Cyanosis.—Not infrequently, intermittent cyanosis may be encountered in infants with primary cardiac tumors and, as a rule, becomes particularly pronounced during periods of crying or while the infant is being fed. In this respect, it may frequently be indistinguishable from the cyanosis encountered in cases of congenital heart disease, vascular ring anomalies, or other extrinsic or intrinsic abnormalities of the trachea or esophagus which produce obstruction. However, in the presence of massive cardiac enlargement resulting in compression and atelectasis of both lungs the possibility of tracheal obstruction being the primary cause of cyanosis is less likely. Furthermore, if cyanosis improves dramatically when the patient is placed in oxygen some of the more severe forms of cyanotic heart disease with a right-to-left shunt may be considered less seriously.

Vomiting and Coughing.—The insidious onset of progressive vomiting associated with severe bouts of coughing, unassociated with a history of aspiration, febrile illness, or a definite pneumonic process in the lungs should suggest the possibility of an abnormality in the position or configuration of the esophagus and trachea. By barium swallow and tracheogram it is possible to outline these structures and reveal extrinsic compression, intrinsic stenosis, or displacement of these organs. In addition, during roentgenologic examination any large cystic structures arising from the mediastinum may be delineated, thus revealing possible pericardial cysts, dermoid cysts, teratomas, or esophageal or gastrointestinal duplications.

Angiocardiography.—Angiocardiography may be carried out though not without considerable risk in those selected cases of rapid progressive cardiac enlargement where all other means of investigation have failed to reveal the cause of the cardiomegaly and the infant has not responded to appropriate medical therapy. It is acknowledged that angiocardiography in such cases will frequently reveal one of the far more common severe congenital abnormalities of the heart.

PROGNOSIS

The prospect of appreciably altering the dismal prognosis of primary cardiac tumors in infancy is not at all encouraging because of their extreme rarity, frequently malignant character, and the difficulty of diag-

nosing them during life. However, it is hoped that routine consideration of cardiac tumors in the differential diagnoses of cardiac abnormalities will lead to earlier and more frequent recognition of this condition and will ultimately permit cures to be achieved in those patients having benign tumors or cardiac neoplasms of low-grade malignancy.

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Medical Progress

A CURRENT SUMMARY OF STRABISMUS IN CHILDREN

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ONE or two out of a hundred children have eyes that are not straight. Four-fifths of these squints are esotropias, that is, the visual axes of the two eyes converge, or "cross." In the adult population there are fewer squints and most of them are divergent. There is a natural tendency, particularly from 6 to 15 years of age, for crossed eyes to tend toward the straight and sometimes on into divergency. Why then is it not fair to say to the parents of a squinting child: "He'll probably grow out of it." Because while the crossing of the eyes may apparently improve without treatment, the child is liable to be left with poor vision in one eye for the rest of his life.

If parents take any action when they notice that a child's eyes are not straight, one with the other, or at least not straight on some occasions, it is often to consult the pediatrician or the general physician. Perhaps the child may be taken to an optometrist, since much of the public does not differentiate medical from nonmedical eye care. Least likely of all to be consulted primarily by the parents of a squinter is the ophthalmologist.

Pediatricians, then, should be provided with a clear and concise concept of the principles of the treatment of the squinting child, so that they may send him on his way without being required to burden themselves with the study of a mass of controversial ophthalmological material, much of it of abstruse nature. Ophthalmologists must shoulder the blame if other physicians find themselves confused in the understanding of the subject of strabismus. The confusion originates from the actively developing nature of our still limited knowledge of strabismus, which is far from a closed subject. Admittedly, "any attempt to fit the complex anomalies of binocular vision into simple clinical classifications for the sake of convenience can only deter the progress of our knowledge of the diagnosis and treatment of these cases." (Dr. Kenneth C. Swan.) While striving for clarification of the pathologic physiology of binocular vision and the successful handling of all or most strabismus cases, we are faced with the squinting child of today for whom we must do everything that is currently possible.

A cardinal point upon which most ophthalmologists agree is that treatment should begin soon after the first crossing of an eye. This flat statement demands some modification, and in the discussion of this point the principles that are well established today as the result of acutal experience are unfolded.

The cogent reason why *every* child suspected of strabismus should have

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a complete eye examination as soon as a squint is noticed is *not* so that every cross-eyed child can immediately begin to receive treatment, but rather that those squinters *who can hope to benefit from treatment* can receive it in time to obtain the maximum benefit, that is, grow into adulthood with two eyes that are straight, two eyes each with good vision, and two eyes that work together completely in a practical, every-day-useful sense. Full binocular function is necessary for perfect depth perception, and it is the only sure way of keeping the eyes straight. Only by the early examination of all squint suspects can those with binocular potentialities be culled from those having practically no chance of achieving binocular vision. We must admit initially that most squinting children—many more than half, including a good many of the operated ones—as handled (or neglected) today never achieve binocular single vision. Many of these can be picked out rather shortly in the first months of observation or even at first examination.

There are numerous "causes" for a strabismus that cannot be hopeful of binocularity. The following are examples of squint situations that commonly preclude an attainment *later* of full binocular single vision: (1) any organic obstacle to good vision in either eye, such as one of the worse degrees of retrolental fibroplasia, (2) permanent paralysis of an extraocular muscle, (3) any squint having a vertical component, (4) any exotropia (divergent squint) that is constantly present, and (5) any squint related to a marked difference in the refractive status of the two eyes. In such squinting children surgery can do nothing more than straighten the eyes cosmetically, and it is wisest to defer this until the last possible moment rather than operate at once. The last possible moment, usually, is just before the child starts to school and comes under the observation of his classmates. We should avoid early operation because eyes that do not see equally well, that have no strong binocular capability and desire, will not stay straight indefinitely, but will wander out of line in time, taking longer to do so the older the patient. These eyes may have to be operated upon repeatedly if this artificial straightness is to be maintained, and if only two operations do the trick for life, one preschool and one in late adolescense, the patient can consider himself fortunate.

A limited expenditure of time and effort should be made for the squinting child of little binocular hope. But measures to ensure good vision in each of the two eyes *separately* should at least be considered, even if function together is not achievable. On the other hand, the treatment of a strabismus with binocular potentialities should include every effort suitable to the particular case, carried out as early as possible. The widely varying opinion as to the value of specific techniques used in the treatment of strabismus, principally occlusion ("patching") and orthoptics ("eye exercises") is due in part to failure to differentiate between cases of squint which have a chance of reaching complete binocular function, and those with no such potentiality. These two types of strabismus should be viewed with differing goals in mind. Nothing less than complete binocular function

should satisfy in cases where it is possible of achievement. Any of the less-than-perfect degrees of cooperation between the eyes has limited value for the visual tasks of daily life. We should be willing to accept cosmetic straightness—a child who appears normal to others but does not enjoy complete normal binocular function—in cases where cosmetic straightness is all that is possible.

Can the functionally hopeful case of strabismus be differentiated from the hopeless ones w i t h o u t proceeding through the full battery of squint treatment techniques to see whether they will succeed or fail?

A personal review of strabismus cases that ultimately reached full binocular single vision* shows, with the rarest exceptions, that (1) good vision was present or developable in *both* of the eyes, (2) the squint had *not* been constantly present from birth, (3) the squint was present only intermittently, or, if constantly present, was reducible (perhaps abolished) by glasses correcting the patient's refractive error.† These are the features common to all ultimately binocular (cured) strabismus cases, although their presence is no guarantee of success. Glasses will sometimes modify the degree of squint only *after* there has been some squint surgery performed, and will not benefit the strabismus before operation. However, it is fair to say that if strabismus is ever to receive total cure it must not appear until some time after birth, and if constant (rather than intermittent), must be modifiable by glasses at an early period. How rare are the exceptions to these requirements? Men having devoted themselves to squint therapy for years, with every adjunctive resource—glasses, occlusion, orthoptics, finished surgical technique—available under their personal commands, are still looking for their first exceptions—with great hope.

Not all pairs of deviating eyes that meet these conditions will reach full binocular function, as there are other factors to be considered in individual cases. But if these are true prerequisites to ultimately successful therapy in strabismus we can defer the cosmetic surgery for many cases and devote our immediate attention to those who have hope of functional cure. Roughly one-fourth of all squints are present at birth, it is estimated, and we can offer these nothing but cosmetic correction at the present state of our knowledge.

Fewer than two-thirds of the children with squints have even a potentiality of binocular single vision, with today's therapy. This theoretical limit derives largely from the fact that the 20 per cent who are exotropic include many cases dating from birth and obviously impossible of functional binocularity, and that among the 80 per cent esotropes about one-third are wholly uninfluenced by glasses.

Simply stated, we can "cure" completely only those squinters who started out normally, "went bad" only later, and who received early treatment. The child we can help is the one who had straight eyes and normal visual oppor-

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†This is not to imply that it is essentially desirable for a child, cross-eyed or otherwise, to possess a refractive error. It merely points up the fact that if a squint has been encouraged by a hyperopia ("farsightedness") it has a good statistical chance of having had nothing worse behind it. It should be remembered that most children with even considerable refractive errors never become squinted. Squinters are themselves a selected group and those related to use of accommodation still more selected.

tunities in his first years of life and whose normal binocular development was interrupted only secondarily by a correctible defect—often a hyperopic refractive error. This type of squint may appear at any time between the ages of 1 and 7 years, and very frequently has its onset at about 18 months when the child uses his accommodation in making his first serious attempts at discriminative vision. Success in treating these patients is proportional to how short a time the interruption—the squint—has been allowed to continue, and how well-established binocular vision was before such interruption. At birth neither the vision of the separate eyes nor the ability to use them together is well developed. At first the eyes wander about a good bit. As the first birthday approaches, vision is becoming more acute and the eyes are beginning to be rotated and used as a pair. By 6 years fusion and vision have been pretty well perfected, and the refractive status is not likely to change much from then on. If a child's eyes are straight for his first three or four years and we can begin treatment shortly after his squint first appears, there is a better than 50 per cent chance of cure. If his eyes were straight only until he was 2, and remained crossed for two or three years before treatment is started, the chances are much poorer.

Unfortunately squints are often present for several years before an eye examination is made, because parents accepted advice to "wait," or feared an operation might be recommended. Not infrequently this neglect itself converts a case in which surgery was originally unnecessary into one where it is now called for. To be perfectly fair the ophthalmologist's correct advice on some occasions will be to "wait and see if the child grows out of it," but this is proper in only a very few cases, most of them being certain intermittent exotropes, whose eyes squint "out," and then only at times, and to only a limited degree. It is rarely the correct advice for esotropes, who squint "in." Since in many cases there is a familial trend to strabismus the siblings of a squinting child should also be examined.

As soon as a squint begins in a child who initially had good sight in both of his eyes, the two eyes will receive separate, differing images simultaneously. The child cannot stand the confusion of two conflicting images, and by a practically automatic mechanism of the brain he learns to ignore the image received by one eye. If he is able to fix with either eye in turn, and only temporarily ignore the other's image, he can maintain good vision in both of his eyes indefinitely. But this "alternator" has only monocular vision always. If a squinter always uses the same eye for fixation he is ignoring the image of the other, that is, he is "suppressing" the vision of the crossed eye. Suppression is usually followed by increasing loss of vision, a functional adjustment to rid the child of an annoying image. This loss of vision in a physically healthy eye due to habitual lack of use is "amblyopia" and if continued for several years (or to 7 or 8 years of age) may become irreversible. Only the central, sharpest vision of the eye is lost, however, apparently because the normal human peripheral vision is not acute enough to cause a conflict. But an adjustment is often made concerning the peripheral vision as well, and the two

eyes develop an abnormal way of making their common judgment of the location of objects around them. This "abnormal retinal correspondence" is an inferior, substitute mechanism forced upon squinting eyes by their lack of straight alignment. In time it becomes permanent, even comfortable by long acceptance, and interference with it may make cosmetic eye surgery on squinting adults treacherous. Abnormal retinal correspondence and suppression are the principal stumbling blocks to the attainment of binocular single vision in children.

The basic principles underlying any treatment of strabismus that has as its goal complete binocular function are but two in number: get the best possible vision in *each* eye, and *keep* the eyes *aligned* by any method whatsoever.

Both must be accomplished at the earliest possible moment because of a third great factor in the development of binocular function upon which we can bring little effective external influence—fusion, the brain's desire to use the two eyes together. Our only recourse to ensure the development of fusion is to accomplish good vision and straight eyes by that period of growth at which nature itself customarily develops fusion. Nature is willing to develop fusion only in a pair of straight, good-seeing eyes and only in the period from 8-12 months to 4-5 years of age, roughly. Our attempts to force fusion at later dates by orthoptic training are miserable by comparison.

To enhance the vision of a poorly seeing eye which has been suppressed we force its use by covering the better seeing eye. The occlusion may have to be alternated from eye to eye and be prolonged for months, if the sight continues to improve. It is not often of lasting value after the age of 6, but a younger child may start visual advancement in a week. Glasses correcting refractive error often improve the effective visual acuity.

Methods of preserving or attaining alignment are surgery, and in some situations, glasses. Surgery shortens or lengthens some of the extraocular muscles, and can do nothing more than change the anatomic positions of the globes in the orbits. Its aim is for the new positions following operation to make it easier for the ocular muscles to move the two globes into parallelism, so that they can the more easily be held in straight alignment by the good visions of the two eyes desiring to work together in good fusion. The selection of the particular muscles to be operated depends on how much correction is needed. It seems evident that both eyes should be operated upon with a symmetrical surgery to achieve best function in all fields of gaze as well as better appearance of the palpebral fissures. Two days of hospitalization are usually sufficient. Operation will bring about half of the esotropias close to straight. The other half will be improved but require further surgery. If functional cure cannot be reached following two operations it probably is unattainable. In about 1 per cent the first operation results in no change or in an overcorrection into divergency. If a squinting child has binocular potentialities surgery should be undertaken as *soon* as the more conservative measures appropriate to the child's age have been given a fair trial without success, or have ceased to give improvement. A squint that is completely abolished by corrective lenses must never be oper-

ated upon in the hope of discarding the glasses; surgery should be intended to correct only that portion of the deviation that is actually anatomical, and not that portion that is physiological.

Glasses can sometimes improve alignment by reducing the accommodation the child may have to exert to overcome a hyperopic refractive error. The excessive convergence efforts that accompany accommodation are simultaneously reduced, and this may straighten the eyes. Glasses as the sole therapy are helpful if amblyopia and abnormal retinal correspondence have not had a chance to develop. A six weeks' trial of corrective lenses is enough to indicate whether a reduction in the squint measurement has been effected. Some children will require bifocal spectacles to keep their eyes straight both for distance and for near use. It should be made clear to parents that the wearing of glasses will not necessarily be permanent. The ultimate goal is for the child to have the weakest corrective lenses that will ensure him both straight eyes and good vision at all times, and for him to be able to go without his glasses on occasion, with the eyes remaining straight.

Orthoptics, commonly known as "eye exercises," can neither directly change alignment nor improve vision. It is a training of the fusion faculty, which is a cerebral activity. It can sometimes promote a normal retinal correspondence or help banish suppression. On occasion the assistance provided by orthoptics will be enough to combine with occlusion, glasses, surgery, or a complex of these in holding the eyes straight. The time of its greatest value is immediately postsurgery. Its principal value is the progressive improvement of binocular functions from lower levels of ability to use the eyes together, to higher levels of cooperation approaching the complete binocular activity of daily life. If this last is achieved orthoptics has been successful; if not, it has been a waste of time. It is by no means worth while in all cases, and experience has shown that there are certain fairly clear prerequisites to its success, such as good vision in each eye, some binocular opportunity earlier in life, eyes anatomically capable of the straight position, cooperation from the child, who must be at least 6 years old, and continued parental support for a series of treatments. In questionable cases a four weeks' trial may be made. Orthoptics must be administered by a technician thoroughly trained in its application, and the physician ordering it must understand its limited indications.

An eye examination of value can usually be made in a very young child. An estimate of visual acuity is made from the baby's ability to fix a light, and from the parents' observations. Children of 3 or 4 years can often give accurate vision determinations with picture charts. The refractive error of an infant 1 year old can be determined objectively with the retinoscope, perhaps with the assistance of Nembutal per rectum. The degree of squint can be compared before and after the use of atropine for several days to reveal any accommodative component.

Glasses are often successfully worn by infants a year old. Sometimes one has more difficulty removing the baby from the spectacles than he experienced connecting the two originally. Patching can be instituted early if the mother gives wholehearted support.

Orthoptics is of diagnostic value as early as the age of 4 years, but not therapeutically until 6 or 7 years at the earliest. Operation on the ocular muscles is feasible at one year; earlier the risk of anesthesia is too dangerous.

The ophthalmologist's insistence on a functional result where it is possible of attainment may only bring him recrimination. Parents are often unwilling to continue treatment once a squint has been reduced to the neighborhood of 5 degrees, giving apparently straight eyes. Binocular perfection may have little more chance here than with the greater angle of squint previously present, unless treatment be continued. Correction that is cosmetic only is the easy way out.

SUMMARY

1. Only the pediatrician, and to a lesser degree the family physician and the optometrist, can change the public conception that expectant treatment is proper for the squinting child.

2. The period of opportunity for development of visual function (birth to age 5 to 6 years) *will* be used, squint or no squint. With strabismus it will be devoted to the perfection of some abnormal, inferior type of two-eyed function, which in time becomes fixed and irreversible—"right" for the particular case.

3. The functional cure of squint—the attainment of binocular single vision—is difficult of realization, and is rarely achieved unless the squinting child is seen early by the ophthalmologist.

4. Functionally perfect straight eyes are not possible for all children. Only some 60 to 65 per cent of squinters have even a potentiality of binocular single vision, as of current methods of treatment.

5. Functional cure demands the presence or creation of similarity and congruity in the binocular mechanism—a similarity of images for fusion and a congruity of muscles for motility. Also necessary is an early and continuing opportunity for this symmetrical binocular machinery to learn to function as a unit.

6. Good vision in each eye is the single most important prerequisite to maintaining a normally straight pair of eyes, or aligning a pair of squinted ones.

7. Orthoptics ("eye exercises") should be hopefully undertaken only in selected strabismus cases. It is rarely of value unless accompanied by other forms of squint therapy as occlusion, glasses, and surgery, and cannot be used until the child has reached an age which of itself makes functional cure of squint unlikely.

9. Unless binocular single vision is potentially achievable, and unless parents, child, and ophthalmologist are ready to expend a maximum, sustained, and often expensive effort to obtain it, it is better to defer a merely cosmetic surgery until kindergarten age. Operations that are merely cosmetic suffer losses of straightness in time.

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Comments on Current Literature

SPREAD OF RICKETTSIALPOX

THE July, 1953, issue of the *Annals of Internal Medicine* carries an article by Nichols, Rindge, and Russell[1] concerning the spread of rickettsialpox. In an entertaining manner these authors present observations which elucidate the relationship between the habits of the house mouse, and of the mite commonly parasitizing the house mouse, and the natural history of this disease. Rickettsialpox, recognized as a disease entity during outbreaks in New York City in 1946,[2] was soon shown to be caused by *Rickettsia akari*, and the mite, *Allodermanyssus sanguineus*, a common ectoparasite of the house mouse, was shown to be responsible for the transmission of the disease to man.

Until a minor outbreak occurred in an apartment house in Hartford, Connecticut, in 1952,[3] only two cases, naturally occurring, had been reported outside the metropolitan limits of New York. These two patients were both resident in Boston.[4] Subsequently four cases occurred in an industrial area in metropolitan Philadelphia.[5] This confinement of the disease to urban districts suggested to the authors that some explanation for its unique distribution might be found in a better understanding of the habits of the house mouse and the mouse mite.

With this purpose in mind, the authors made a careful analysis of the habits and activities of house mice as recorded in two extensive investigations which were carried out for purposes of pest control.[6, 7] One of these investigations involved 1,572 house mice, a number determined by identification and multiple trapping over a period of eight months, food supply being constant and readily available in the regions studied. Seventy per cent of these mice identified did not move out of an area of about ten feet; 90 per cent confined their activities to an area of thirty feet. Only one mouse, "the Dr. Livingston of the group, extended his travels to 140 feet on one expedition, the maximal range recorded." On the basis of these observations it appeared that mice do not venture far afield, if food is plentiful and housing satisfactory. Mice also seem to have considerable adaptability to varying conditions and temperature range, and in the American urban environment the only significant enemy of the house mouse seems to be the rather fierce Norway rat, whose threat is chiefly one of food piracy. Where food is not readily available, mice have been known to make nightly excursions to wheat fields as distant as one-half mile from the home base.[7]

Nichols, Rindge, and Russell sought to correlate such ecologic observations concerning the house mouse with some of the gross variations in the case distribution of rickettsialpox in New York City. As of July, 1952, 724 cases of rickettsialpox had been reported to the New York City Health Department: 1946, 174 cases; 1947, 157 cases; 1948, 135 cases; 1949, 105 cases; 1950, 86 cases; 1951, 55 cases; and in 1952, 12 during the first six months. The authors compare the case distribution over the intervening years with that given by Greenberg[2] for the 1946 outbreak. In 1946, cases centered in lower Manhattan, east and west of Central Park and in cer-

tain sections of the Bronx. The occurrence of cases from time to time in Manhattan and the Bronx during the past six years indicates continued activity and extension of the endemic areas represented by the cases in 1946, particularly in northwest Manhattan Island. During the same period comparatively few cases have been reported in Brooklyn and Queens, only 36 cases, the majority occurring at widely separated points during a one-year period. At the original epidemic location in Kew Gardens, 26 cases have developed subsequently, suggesting significant continuity of intermediate host infection. Greenberg[8] has attributed this recurrence in Kew Gardens to a probable reintroduction of susceptible hosts into the environment.

The authors do not believe that differences in population density account for these rather striking variations in the behavior of the disease in the Manhattan-Bronx and the Brooklyn-Queens sections of the city. Pressures of increasing population have been about comparable in the two areas. Differences in the type of housing, however, seem significant to them. In the Brooklyn-Queens area, housing still consists essentially in s m a l l e r, separated dwellings in which a given mouse population would tend to remain relatively confined, offering less continuity of mouse reservoir of the disease. In the Manhattan-Bronx section, however, wall-to-wall type of housing would provide for the mouse populations ready access to all units within the block, with probable extension of mouse reservoir.

A significant observation which has been quite consistent over the six-year period is that of the relatively few scattered cases recorded for the poorer slum section of Manhattan and the Bronx, as compared with the greater concentration of cases in the middle and upper income bracket areas. This apparent paradox seems to be explained to the authors' satisfaction by the prevalence of the large Norway wharf rat in the poorer slum areas, where profusion of garbage and litter affords ideal conditions for the growth and multiplication of rats. That rat populations are considerable in these parts of the city is emphasized by the fact that some 300 rat bites in human subjects are reported each year in these sections. It is interesting that this traditional competitor of the house mouse is quite efficient apparently in eliminating mice from a community. In pest-control campaigns in these areas, 1,800 separate premises have been investigated for rats. The Norway rat comprised the great majority of the rodent population, mice being trapped in conjunction with rats in only six instances. Should the rat mite become a natural vector[9] in rickettsialpox, it is conceivable that the epidemiologic picture might be altered significantly.

Since the mouse mite, *Allodermanyssus sanguineus,* requires a moist, relatively warm environment, housing conditions which permit low temperatures to prevail, or fall to extremes during the night, tend to retard its multiplication. Such conditions of living are found more commonly in slum areas.

An analysis of the Hartford, Connecticut, outbreak of rickettsialpox seems to bear out some of the general impressions gained from the New York studies. The site of this outbreak was a fashionable Tudor structure of three stories, consisting of six well-kept, r a t h e r handsomely furnished, seven- or eight-room apartments, housing a total of thirteen adults. This building and a similar companion structure, twenty yards distant, which was also found to be mouse-infested, but whose occupants showed no signs of the disease, were surrounded by ample well-kept lawns, shrubbery, and gardens. In January, 1952, eight of the thirteen occupants of the one building fell ill with rickettsialpox. A survey of the two structures indicated that mice had access to the incinerators in which

adequate food supply was present almost continuously. At the time of the outbreak it was noted that the outer walls of the incinerators were covered with mites, some of which were engorged with blood, and some apparently unfed. A collection of these mites from the incinerator walls yielded an infectious agent identified as *Rickettsia akari*. The conditions in both apartment structures were such as to provide for maximal association of mice at a site most favorable for the multiplication of *A. sanguineus*.

In a summary of these epidemiologic studies the authors state that at the present time, rickettsialpox enjoys its widest distribution and greatest activity in the middle and upper income sections of cities where density of housing is great. The disease is less prevalent in rat-infested slum areas, and is of relatively rare occurrence in those parts of a city where separated smaller dwelling units prevail.

RUSSELL J. BLATTNER

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News and Notes

The following were certified by the **American Board of Pediatrics**, at the examination at Miami, Fla., Oct. 9, 10, and 11, 1953.

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The following are schedules for the coming examinations to be held by the Board:

Written examinations at selected places will be held on Friday, Jan. 15, 1954. This is the only written examination which will be given during 1954.

The oral examinations will be:

Indianapolis, Ind., Dec. 4, 5, and 6, 1953.
Washington, D. C., Feb. 19, 20, and 21, 1954.
Des Moines, Iowa, March 19, 20, and 21, 1954.
New York, N. Y., May 1, 2, and 3, 1954.
San Francisco, Calif., June 25, 26, and 27, 1954.
Chicago, Ill., Oct. 8, 9, and 10, 1954.
New Haven, Conn., December, 1954.

At the 22nd Annual Meeting of the **Academy of Pediatrics** in Miami on October 8, Dr. Crawford Bost of San Francisco was elected President-Elect. Dr. Roger L. J. Kennedy of Rochester, Minn., President-Elect, took over the presidency for the coming year, succeeding Dr. Philip S. Barba.

Over 1,000 were registered at the **Seventh International Pediatric Congress** at Havana, October 12 to 17. More than twenty-five countries were represented. The next Congress which will take place in 1956 will be held at Copenhagen, Denmark. Prof. P. Plumb of Copenhagen was elected President for the Eighth Congress, and Prof. G. Fanconi of Zurich was re-elected Secretary.

The **University of Minnesota** will present a continuation course in Pediatrics for general physicians Jan. 7 to 9, 1954, at the Center for Continuation Study. Recent advances in the management of common pediatric problems will be stressed. Care of the premature infant will be discussed, and the

therapy of burns will be taken up along with many other topics of general interest. The course will be presented under the direction of Dr. Irvine McQuarrie, Professor and Head, Department of Pediatrics at the University of Minnesota. Lodging and meal accommodations are available at the Center for Continuation Study.

A one year's fellowship in Pediatric Neurology is available in the **Julian D. Levinson research foundation** in conjunction with the Children's Division of the Cook County Hospital. Salary $3,500.00. For further details communicate with Dr. Abraham Levinson, Children's Division, Cook County Hospital, Chicago 12, Ill.

Book Reviews

Developmental Psychology. Elizabeth B. Hurlock, Ph.D., New York, 1953, McGraw-Hill Book Co., 556 pages. Price $6.00.

While Hurlock's new text covers the stages of development from birth through old age, by far the larger part discusses the development during the age periods with which the pediatrician is concerned, as infancy, babyhood, early and late childhood, puberty, and early adolescence. Dr. Hurlock has presented a mass of well-selected material in an orderly and readable form. She has drawn on medical sources as Aldrich, Gesell, McGraw, and others with which the pediatrician is fairly familiar, and, in addition, the huge volume of psychologic studies and literature which is largely beyond his ken. Much of this is not only of interest but of great importance to workers in the field of childhood. It is a book that the practicing pediatrician can browse through with profit, and one that can be recommended for careful reading by the younger pediatrician who is preparing for his Board examination. While not primarily a text for the physician or medical student, it is a book that is most useful for the physician who has selected childhood as his special field of work, which implies and necessitates a knowledge and understanding of children as well as disease. From this standpoint it deserves a place in the pediatric library.

Health Services for the Child. Edward R. Schlesinger, M.D., New York, 1953, McGraw-Hill Book Co., 403 pages. Price $7.50.

This is a text by the Associate Director of Medical Services, and formerly Director of Maternal and Child Health Services, of the New York State Department of Health. It is primarily a text for physicians, with the purpose of giving an integrated picture of health services for mothers and children, rather than a manual for public health workers. After a group of chapters considering such basic matters as community programs, administrative relationships, and professional personnel, the essential health services are considered—mental, dental, child safety, etc. A third section considers the details of health supervision, and a final section discusses the special problems of children with various handicaps. There is a good bibliography following each discussion. The book will be of value and interest to pediatricians who are called upon to take an active part in health programs, either as participating personnel or as Board or Committee members.

Speech Problems of School Children. Chicago, 1952, The National Society for Crippled Children and Adults, 60 pages. Price $1.25.

This is a symposium on speech problems arranged by the Society for Crippled Children and Adults, the American Psychological Association, and the American Speech and Hearing Association. In all, seven phases are presented by workers in this field, including the development of speech and language in children, organic speech disorders, hearing disorders, and training methods. The symposium gives a rapid survey of speech problems and the methods of diagnosis and training in present use.

Editor's Column

BORIC ACID

IT IS incredible that a drug with such toxic propensities and doubtful therapeutic value should continue to enjoy such popularity in the armamentarium of so many physicians and occupy such a prominent place in the home medicine cabinets. Perhaps the explanation of this paradox is that advanced by Drs. Goldbloom, that many topical agents survive as therapeutic agents, not so much in their power to do good, but in their failure to do harm. Apart from accidental poisoning with boric acid there should be greater awareness on the part of the medical profession and laity of the toxic effects of absorption of boric acid from broken skin surfaces and mucous membranes. The articles by Drs. Goldbloom and Ducey and Williams in this issue of the JOURNAL add to an imposing list of documented medical testimony as to its potential toxicity.

Boric acid should be replaced in medical practice with more efficient and safer medication, and pharmaceutical houses should cooperate in an educational campaign to acquaint the public of its potential danger. Perhaps it is not by mere rhetorical coincidence that the major source of the boron compounds comes from Death Valley.

H. G. P.

SHOULD THE CHILD WITH CONVULSIVE DISORDERS BE IMMUNIZED AGAINST PERTUSSIS?

IT IS well established that encephalopathies may follow various types of immunization procedures as smallpox vaccination. The exact mechanism is uncertain, but there is general agreement that a susceptible soil plays an important part. In 1949 Byers and Moll[1] focused attention on pertussis immunization in this connection by reporting a group of fifteen children observed at the Children's Hospital in Boston who had developed cerebral symptoms following the injection of pertussis vaccine. Isolated instances had been reported previously. No one vaccine product was responsible. In the same report they also stated that the records of the hospital showed twenty-six cases of encephalopathy developing during an attack of pertussis. Half of these were in infants under 7 months of age. They raised the point as to whether universal pertussis vaccination prevented encephalopathies or death from pertussis in large groups, and if this were not true, but immunization simply led to the avoidance of the inconvenience of the disease, whether or not the risk involved was considerable. This report led Toomey[2] to investigate by questionnaire the experience of others. He obtained reports on thirty-eight cases in which convulsions had

followed the injection of pertussis vaccine, and drew the conclusion that no child should receive injections of pertussis vaccine in large amounts when there was a family history of convulsions, a present history of convulsions, or an illness of any kind. Miller,[3] the following year, in leading a Round Table discussion on immunization, stated that he felt children with cerebral damage should not be given pertussis vaccine, and that children with histories of febrile convulsions should be inoculated first with a fractional dose. Cockburn,[4] in an English symposium on immunization, stated that he felt it unwise to inoculate with pertussis vaccine any child who has a personal or family history of convulsions. These last three represent what might be termed the immunologist's viewpoint.

So far as we can determine by questioning a number of pediatricians, the present tendency among practitioners is not to use pertussis vaccine if a family or personal history of convulsions is known. As to current teaching textbook statements, Bradford,[5] in his discussion of pertussis in *Mitchell-Nelson* (1950), does not go into the matter. In his more recent discussion in *Holt-McIntosh* (1953) he states that he considers the risk a minimal one that should be taken. Hodes,[6] in a discussion of pertussis encephalopathy in this text, considers that the incidence of encephalopathy following pertussis immunization is so low that it leaves no doubt regarding the wisdom of continuing mass pertussis immunization programs. He interprets existing data as indicating the immunization should not be continued if symptoms referable to the central nervous system develop.

In a recent discussion before the American League Against Epilepsy in Boston in August, 1953, which will be found on page 652 of this issue, Dr. Melin, of the Clinic for Convulsive Diseases, Stora Sköndal, in Stockholm, raises an interesting and important question. As the result of his experience he feels the danger to a child with a convulsive tendency or background from an attack of pertussis is greater than the danger of an encephalopathy resulting from the immunization of such a child with pertussis vaccine. Dr. Melin's approach is from the standpoint of the seizured child, rather than the immunological.

As the attitude and experience of physicians working with seizured children is probably more important in regard to the question raised than the viewpoint of the immunologist, several were asked to comment on Dr. Melin's contention.

Dr. Peterman of Milwaukee wrote as follows:

> In answer to your letter of September 3, re the paper by Dr. Melin, may I say this: I agree entirely with the author. In 30 years of practice with a very large number of pertussis immunizations, I have seen no encephalitis or encephalopathy except in six cases following the use of a combined pertussis vaccine and diphtheria toxoid. In the same period, I have seen a number of cases of brain damage, particularly hemorrhage, following pertussis in children who have not been immunized with the vaccine. I have never hesitated in the least to give any child with a history of convulsions the necessary protection against pertussis.

Dr. Samuel Livingston, Chief of the Epilepsy Clinic at the Johns Hopkins Hospital, comments as follows:

> I agree with Dr. Melin's recommendation that prophylactic pertussis immunizations be administered to children with convulsive disorders. In my opinion, the

value of pertussis vaccine in the prevention of whooping cough greatly outweighs the likelihood of the development of the "encephalopathies" which have been reported to have occurred in association with pertussis inoculations. In fact, I question whether children with convulsive disorders or "convulsive tendencies" are any more likely to develop cerebral complications from pertussis immunizations than are other children. I base my opinion on the following observations.

During the past 18 years I have had the opportunity to study approximately 7,000 epileptic children. In 11 of these patients, epilepsy developed following routine prophylactic immunizations. Two of these 11 patients developed epilepsy following encephalomyelitis which complicated smallpox vaccinations. Two patients developed epilepsy following prophylactic treatment against rabies, and 7 began having recurrent seizures soon after the administration of pertussis immunizations. The clinical course of these latter 7 patients was quite similar to those reported by Byers and Moll. None of these 11 patients had suffered with convulsions of any type prior to the administration of prophylactic immunizations. There was no history of epilepsy or allied disorders in the families of these patients.

Six hundred eighty-six of our patients with cryptogenic (idiopathic) epilepsy had received routine prophylactic pertussis immunizations between the ages of 3 months and one year. The symptoms of cryptogenic epilepsy did not appear in any of these 686 patients until after the third year of life. Several of these patients suffered with a short febrile generalized seizure at the time of the initial pertussis inoculation, but none of them presented any other significant cerebral symptoms. Two hundred ninety-eight of these 686 patients with cryptogenic epilepsy also received at least one booster pertussis inoculation after the third year of life at which time they had already presented symptoms of cryptogenic epilepsy. Again, in none of these cases were there any significant reactions. One could certainly assume that these 686 patients possessed a "predisposition or tendency to epilepsy" at the time they were given their initial prophylactic pertussis immunizations.

Prophylactic pertussis immunizations are most often given between the ages of 3 months and one year. During this period of life, the most common convulsive disorders are those associated with extracranial infections such as acute tonsillitis, otitis media, etc. (simple febrile convulsions), and those associated with congenital cerebral defects or cerebral birth injuries (secondary or organic epilepsy).

Ninety-six of our patients who had suffered with simple febrile convulsions were given pertussis inoculations. Ten of these patients had a recurrence of simple febrile convulsions soon after the initial pertussis inoculation was administered. All of these ninety-six patients have been observed for a period of 10 years or longer. None of them have, as yet, developed epilepsy.

Two hundred eighty-four of our patients with secondary epilepsy, who began having seizures between 3 months and one year of age, were also given routine pertussis immunizations. All of these patients had been having rather frequent epileptic seizures at the time the inoculations were given. In a few instances there was a temporary increase in frequency or severity of seizures following the pertussis inoculations, but in no case were any apparent permanent effects encountered. We have also observed many hundreds of other children who suffered with similar secondary convulsive disorders who were not given pertussis inoculations. The course of the epilepsy was essentially the same in both groups.

In conclusion, I would like to state that there is no doubt that some children have sustained permanent cerebral damage as a result of pertussis immunizations. I do not, however, believe that one can predict which child is more likely to be adversely affected by pertussis inoculations any more than one can predict which epileptic patient will develop a pancytopenia as a result of Tridione or Mesantoin therapy. Our observations certainly indicate that a history of convulsions or even an active convulsive disorder does not necessarily contraindicate the administration of pertussis inoculations. In view of the frequency and severity of complications of whooping cough in children, particularly the very young child, I believe that one has more to gain by giving routine prophylactic per-

tussis immunizations than by omitting them. Again, in view of the fact that pertussis is known to adversely affect the central nervous system of children with especial severity, there is a much greater chance of increasing the epileptic population by allowing a child to contract pertussis than by the administration of pertussis inoculations.

Dr. William Lennox, of The Neurological Institute of the Children's Medical Center in Boston, commented as follows:

Dr. Melin's observations are timely and his conclusions sensible, although "recommendation" of vaccination for epileptic children seems too strong a word. Convulsions are not as much feared as cerebral pathologic lesions. Hence, an existing lesion or an allergy to immunizing agents should carry a greater warning than a possible lowered threshold to convulsions. I have not advised against immunization of otherwise healthy children having genetic epilepsy. However, I seem especially to remember convulsions that have followed prophylactic injections. This may be because complications that are doctor, rather than nature, induced remain more vividly in the memory. If a convulsion follows an initial injection, common sense condemns subsequent injections or judge the following. Monozygotic twins were born prematurely. When 6 weeks old, because pertussis was prevalent, they were given pertussis vaccine and both went into prolonged status epilepticus and probable chronic epilepsy.

Experiences such as Dr. Melin's need to be multiplied before we can judge whether pertussis or its attempted prevention carries the greater danger. The problem is three-sided. What is the incidence of pertussis in vaccinated as compared with unvaccinated children? What is the incidence of convulsions following pertussis on one hand and following attempted immunization on the other hand? A fourth side should be added —How do the neurological or mental sequelae (with or without convulsions) compare? Dehydration, cerebral venous congestion, and vascular thrombosis may attend pertussis, and diffuse encephalopathy the allergic or toxic reactions to vaccine. Even one such case in a thousand is too many. More study is greatly to be desired.

Dr. Randolph Byers of the Children's Medical Center in Boston, whose report in 1949 drew attention to the problem, made the following comment:

I have your letter of September 17, and I have read the report by Melin. From my point of view I should say he had followed very much the procedure that I have usually followed in relation to children who seem defective to me.

I am quite sure that pertussis is more of a danger to these children than pertussis vaccine, and I have usually sat down with the mothers and explained the dilemma in which we found ourselves and told her that the vaccine might produce the convulsions, but that in my opinion the chances that the pertussis itself would damage the child was considerably greater. In almost all instances the mothers have wanted to have their children vaccinated, and I have vaccinated them.

When I have had a child who seemed defective to me and who had no brothers or sisters and to whom pertussis was not likely to be brought, I have delayed the use of the vaccine until the child was active enough to get out and run a chance of contracting the disease. Whether this latter makes any sense or not I haven't any idea.

In the follow-up studies on pertussis in infancy made by Byers and Rizzo,[7] the following conclusion was reached:

"Finally, since pertussis vaccine seems prophylactically efficacious, the risks of its use in infancy are probably outweighed by those of the disease itself."

So the matter stands, and it puts the practitioner on the horns of a dilemma. If he decides to immunize a child with a personal or family history of convulsive disorders there is the possibility of its causing convulsions and even an irreversible encephalopathy, for which, he realizes full well, he will in all probability be blamed. On the other hand, if he

withholds, he takes and assumes a deliberate risk, as pertussis in a child with convulsive tendencies not infrequently is complicated by an encephalopathy. Perhaps some peace of mind can be gained, if he withholds, by the fact that immunization of all children with whom the child with convulsive tendencies comes in contact gives a certain degree of protection, as the risk of contracting pertussis goes down in proportion to the percentage of the population which is immunized. For one decision he has the backing of some of the immunologists and for the other that of experienced workers in the field of seizured children. It is not an easy decision, as the average practitioner is in a somewhat different position than the specialist. If he does decide to immunize, the situation should be explained to the parents and the immunization started cautiously with fractional doses. It does not seem in the present state of our knowledge and conflicting viewpoints that malpractice can be charged if there is an unfortunate ending to either choice. It is the time-honored situation of "I'll be damned if I do and damned if I don't."

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A REPORTED INCREASE IN ACCIDENTAL POISONING IN CHILDREN IN SCOTLAND

IN A recent joint study and report,* Craig of Edinburgh and Fraser of Aberdeen note an increase in the accidental poisoning rate in the child population of their cities since 1948. This was due to a sharp rise in the number of cases of poisoning due to medicaments, as aspirin, barbiturates, and ferrous sulfate. The household accident rate from burns and scalds has remained steady and linear. They call attention to the fact that this abrupt rise in medicinal poisoning occurred at about the same time as the introduction of the National Health Service, but are unwilling to assume that it was entirely a matter of cause and effect. They note that "prescribing" has doubled in Scotland since 1948, but that medicinal poisoning in children has trebled. From their figures we note that poisoning from barbiturates is higher in Scotland than in England, the incidence of iron poisoning is about the same, and poisoning from strychnine is more frequent in England. This fits in with reports from other countries which tend to show that the causes of accidental poisoning vary according to local customs and conditions.

*Craig, J. O., and Fraser, M. S.: Arch. Dis. Childhood 28: 259, 1953.

INDEX TO VOLUME 43

AUTHORS INDEX*

A

ABELLA, PABLO U., The use of the Gardner-Murphy pediatric scalp-vein infusion outfit, 709

ABRAMS, MORRIS (WITH WATTENBERG, CARL A., LEWIS, H. KENT, AND LEWIS, JOHN), Bilateral epididymal tuberculosis in an infant, 443

ABRAMSON, HAROLD, GREENBERG, MORRIS, AND MAGEE, M. CATHERINE, Poliomyelitis in the newborn infant, 167

ADAMS, FORREST H. (WITH ANDERSON, RAY C.), Congenital paroxysmal tachycardia, 668

ANDERSON, FRANK M., AND CARSON, MERL J., Spinal cord tumors in children, 190

ANDERSON, RAY C., AND ADAMS, FORREST H., Congenital paroxysmal tachycardia, 668

B

BACALA, J. C., AND BURKE, R. J., Generalized cytomegalic inclusion disease, 712

BAKWIN, HARRY, The early development of children with schizophrenia, 217

—, AND BAKWIN, RUTH M., Homosexual behavior in children, 108

BAKWIN, RUTH M. (WITH BAKWIN, HARRY), Homosexual behavior in children, 108

BAUMANN, FRANZ, PEARSON, DELBERT E., AND LEVIN, MARCIA, Adrenal cortical steroids in the management of a case of meningococcemia, 575

BEDO, ANDREW V. (WITH ODESSKY, LOUIS, JENNINGS, KENNETH G., SANDS, IRVING J., ROSENBLATT, PHILIP, WEISLER, HARRY, AND NEWMAN, BENJAMIN), Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. I. A clinical study of forty-one cases with follow-up studies, 536

—(WITH ODESSKY, LOUIS, ROSENBLATT, PHILIP, JENNINGS, KENNETH G., SANDS, IRVING J., WEISLER, HARRY, AND NEWMAN, BENJAMIN), Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. II. Cerebrospinal fluid and serum studies in measles encephalitis and encephalomyelitis with special reference to phosphorus, cholesterol, and magnesium, 695

— (WITH ODESSKY, LOUIS, WEISLER, HARRY, AND NEWMAN, BENJAMIN), Encephalitis in children with measles who were given attenuating doses of gamma globulin, 404

— (WITH SMITH, EMIL, AND ROSENBLATT, PHILIP), Sickle-cell anemia crisis, 655

BERGER, S. M., BOREADIS, A. G., AND KREMENS, V., Bronchial adenoma, 417

BERNSTEIN, SIDNEY, AND KANOF, ABRAM, Rehabilitation of a child with numerous congenital defects, 434

BERREY, BEDFORD H., AND DANNENBRING, F. G., An unusual form of central nervous system disease in a newborn infant, 388

BIVINGS, LEE, Gamma globulin dosage and measles control, 401

BLATTNER, RUSSELL J., Histoplasmosis, 489

—, Infantile eczema, 359

—, Infection in newborn baby, 112

—, Poliomyelitis: incidence of subclinical infection during an epidemic, 620

—, Smallpox vaccination and pregnancy, 229

—, Spread of rickettsialpox, 740

BLOCH, HARRY (WITH TURIN, RICHARD, AND KAZNETZ, JOSEPH), Hematemesis: report of a case in an infant due to arterial bleeding from the base of the tongue, 96

BOREADIS, A. G. (WITH BERGER, S. M., AND KREMENS, V.), Bronchial adenoma, 417

BORENSTINE, JOSEPH, AND PRINTZ, JOSEPH H., Meconium ileus (complete obstruction) in a premature infant, 208

BRAZELTON, T. BERRY, HOLDER, RICHMOND, AND TALBOT, BEATRICE, Emotional aspects of rheumatic fever in children, 339

BROWN, H. W. (WITH CHAN, K. F.), The treatment of pinworm (Enterobius vermicularis) infection with bacitracin and Sulfasuxidine, 290

BROWN, SPENCER F., Effect of penicillin injections during acute poliomyelitis, 174

BRUTON, OGDEN C. (WITH JOHNSON, ELOISE EBERLING), Surgically acquired hypothyroidism in a child with coarctation of the aorta, 449

BUNDY, WALTER E. (WITH HENNIGAR, GORDON R., THABET, RAYMOND, AND SUTTON, LEE E., JR.), Salmonellosis complicated by pancarditis, 524

*July, pp. 1-120; August, pp. 121-236; September, pp. 237-370; October, pp. 371-498; November, pp. 499-630; December, pp. 631-750.

BURGER, ROBERT A. (WITH MARCUSE, PETER M., AND SALMON, GEORGE W.), Hamartoma of the hypothalamus, 301

BURKE, R. J. (WITH BACALA, J. C.), Generalized cytomegalic inclusion disease, 712

C

CAMPOS, JOSEPH (WITH KNAUS, WILLIAM E., AND ROSE, WILLIAM), Meigs' syndrome, 88

CARSON, MERL J., AND MONTGOMERY, THEODORE A., Congenital hyperhidrosis: therapy with Hydergine and Banthine, 274

— (WITH ANDERSON, FRANK M.), Spinal cord tumors in children, 190

CHAN, K. F., AND BROWN, H. W., The treatment of pinworm (Enterobius vermicularis) with bacitracin and Sulfasuxidine, 290

CHANDLER, FREMONT A. (WITH LIDGE, RALPH T.), Charcot-Marie-Tooth disease, 152

CHICK, FORRIS E. (WITH MASCIA, ARMOND V., AND LEVY, WALTER E.), Effects of rubeola on tuberculosis, 294

CHIGIER, E., Three unusual cases of meningitis in children, 54

CLEMONS, HELEN (WITH SIFONTES, JOSÉ E., WILLIAMS, R. D. BROOKE, AND LINCOLN, EDITH M.), Observations on the effect of induced hyperglycemia on the glucose content of the cerebrospinal fluid in patients with hydrocephalus, 243

CLIFFORD, STEWART H., Post hoc, ergo propter hoc, 237

COHEN, ROBERT, O'CONNOR, ROBERT E., TOWNSEND, THOMAS E., WEBB, PATRICIA ANN, AND MCKEY, ROBERT W., Western equine encephalomyelitis, 26

CRISMON, CATHRINE S. (WITH GREULICH, WILLIAM WALTER, AND TURNER, MARGARET L.), The physical growth and development of children who survived the atomic bombing of Hiroshima or Nagasaki, 121

CRUICKSHANK, WILLIAM M. (WITH FREED, EARL X.), The relation of cardiac disease to feelings of fear, 483

D

DAESCHNER, C. WM., SALMON, GEORGE W., AND HEYS, FLORENCE M., Cat-scratch fever, 371

DANNENBRING, F. G. (WITH BERREY, BEDFORD H.), An unusual form of central nervous system disease in a newborn infant, 388

DAVIDSON, WILLIAM D., A brief history of infant feeding, 74

DESMOND, MURDINA M., Observations related to neonatal hypoglycemia, 253

DONALDSON, J. S., SANKEY, H. H., GIRDANY, B. R., AND DONALDSON, W. F., Osteochondroma of the distal femoral epiphysis, 212

DONALDSON, W. F. (WITH DONALDSON, J. S., SANKEY, H. H., AND GIRDANY, B. R.), Osteochondroma of the distal femoral epiphysis, 212

DOYLE, P. J., AND LIVINGSTON, SAMUEL, The use of Mysoline in the treatment of epilepsy, 413

—, —, AND PEARSON, PAUL H., Use of Milontin in the treatment of petit mal epilepsy (three per second spike and wave dysrhythmia), 164

DUCEY, JAMES, AND WILLIAMS, D. BROOKE, Transcutaneous absorption of boric acid, 644

DUSHAN, THEODORE A. (WITH KREIDBERG, MARSHALL B.), Paroxysmal auricular tachycardia associated with Wolff-Parkinson-White syndrome in a newborn infant, 92

E

EARLE, ALEXANDER, M., Leukemia in American Samoa, 398

F

FLETCHER, MARY CRITZ, The developing fundus oculi of the premature infant and its relationship to retrolental fibroplasia, 499

FORTUNATO, J. (WITH KLEIN, R., AND PAPADATOS, C.), Serum corticoids in children under stress, 385

FREED, EARL X., AND CRUICKSHANK, WILLIAM M., The relation of cardiac disease to feelings of fear, 483

FREEMAN, MAX E., AND PARKER, GEORGE F., Treatment of staphylococcic pericarditis with bacitracin, 720

G

GEDDES, A. K., AND MOORE, S., Acute (infantile) Gaucher's disease, 61

GIRDANY, B. R. (WITH DONALDSON, J. S., SANKEY, H. H., AND DONALDSON, W. F.), Osteochondroma of the distal femoral epiphysis, 212

GOLDBLOOM, ALTON (WITH GOLDBLOOM, RICHARD B.), Boric acid poisoning, 631

GOLDBLOOM, RICHARD B., AND GOLDBLOOM, ALTON, Boric acid poisoning, 631

GOULD, WILBUR J. (WITH MAIER, HERBERT C.), Agenesis of the lung with vascular compression of the tracheobronchial tree, 38

GREENBERG, MORRIS (WITH ABRAMSON, HAROLD, AND MAGEE, M. CATHERINE, Poliomyelitis in the newborn infant, 167

GREULICH, MILDRED L. (WITH GREULICH, WILLIAM WALTER, CRISMON, CATHRINE S., AND TURNER, MARGARET L.), The physical growth and development of children who survived the atomic bombing of Hiroshima or Nagasaki, 121

GREULICH, WILLIAM WALTER, CRISMON, CATHRINE S., AND TURNER, MARGARET L., WITH TECHNICAL ASSISTANCE OF GREULICH, MILDRED L., AND OKUMOTO, YOSHIO, The physical growth and development of children who survived the atomic bombing of Hiroshima or Nagasaki, 121

GROVE, FRANCES, E. (WITH WARNER, MARY P.), A study of forty cases of ringworm of the scalp, 267

GRUNWALDT, EDGAR (WITH HARTMANN, ALEXIS F., AND JAMES, DAVID H., JR.), Blood galactose in infants and children, 1

H

HADJIMARKOS, D. M., Dental caries among elementary school children in Oregon, 393

HARRIS, IRWIN L. (WITH SMITH, EMIL, AND ROSENBLATT, PHILIP), Acute poliomyelitis: clinical and statistical study of 263 cases, 9

HARTMANN, ALEXIS F., GRUNWALDT, EDGAR, AND JAMES, DAVID H., JR., Blood galactose in infants and children, 1

HENNIGAR, GORDON R., THABET, RAYMOND, BUNDY, WALTER E., AND SUTTON, LEE E., JR., Salmonellosis complicated by pancarditis, 524

HEYS, FLORENCE M. (WITH DAESCHNER, C. WM., AND SALMON, GEORGE W.), Cat-scratch fever, 371

HOLDER, RICHMOND (WITH BRAZELTON, T. BERRY, AND TALBOT, BEATRICE), Emotional aspects of rheumatic fever in children, 339

HOWARD, FRED H. (WITH IVY, ROBERT E.), Sickle-cell anemia with unusual bone changes, 312

HSU, HAN-KUANG, AND SZYPULSKI, JOHN T., Some problems in the diagnosis and treatment of pulmonary tuberculosis in childhood, 661

HUFFMAN, WILLIAM L., PERSKY, LESTER, AND JOELSON, JAMES J., Extravesical ureteral ectopia, 407

I

IVY, ROBERT E., AND HOWARD, FRED H., Sickle-cell anemia with unusual bone changes, 312

J

JAMES, DAVID H., JR. (WITH HARTMANN, ALEXIS F., AND GRUNWALDT, EDGAR), Blood galactose in infants and children, 1

JENNINGS, KENNETH G. (WITH ODESSKY, LOUIS, BEDO, ANDREW V., SANDS, IRVING J., ROSENBLATT, PHILIP, WEISLER, HARRY, AND NEWMAN, BENJAMIN), Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. I. A clinical study of forty-one cases with follow-up studies, 536

— (WITH ODESSKY, LOUIS, BEDO, ANDREW V., ROSENBLATT, PHILIP, SANDS, IRVING J., WEISLER, HARRY, AND NEWMAN, BENJAMIN), Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. II. Cerebrospinal fluid and serum studies in measles encephalitis and encephalomyelitis with special reference to phosphorus, cholesterol, and magnesium, 695

JOELSON, JAMES J. (WITH HUFFMAN, WILLIAM L., AND PERSKY, LESTER), Extravesical ureteral ectopia, 407

JOHNSON, ELOISE EBERLING, AND BRUTON, OGDEN C., Surgically acquired hypothyroidism in a child with coarctation of the aorta, 449

JOSEPH, LESTER G. (WITH MARKS, DENNIS N., AND SHAPIRO, ROBERT S.), Roentgenographic diagnosis of coarctation of the aorta in a two-day-old infant, 453

K

KANOF, ABRAM, AND BERNSTEIN, SIDNEY, Rehabilitation of a child with numerous congenital defects, 434

KARELITZ, SAMUEL (WITH LALL, SEETA), The treatment of diphtheria and diphtheria carriers with terramycin, 35

KAZNETZ, JOSEPH (WITH TURIN, RICHARD, AND BLOCH, HARRY), Hematemesis: report of a case in an infant due to arterial bleeding from the base of the tongue, 96

KEAN, B. H., The treatment of enterobiasis (oxyuriasis). The need for a special sleeping garment, 47

KEEFER, GEORGE P., AND MOKROHISKY, JOHN F., Lead poisoning: roentgenograms of the chest as an aid in diagnosis, 146

KENDIG, EDWIN L., JR., OWNBY, RALPH, JR., TREVATHAN, G. EARL, AND SUTTON, LEE E., JR., The treatment of tuberculous meningitis in children, 532

KLEIN, R., PAPADATOS, C., AND FORTUNATO, J., Serum corticoids in children under stress, 385

KNAUS, WILLIAM E., CAMPOS, JOSEPH, AND ROSE, WILLIAM, Meigs' syndrome, 88
KREIDBERG, MARSHALL B., AND DUSHAN, THEODORE A., Paroxysmal auricular tachycardia associated with Wolff-Parkinson-White syndrome in a newborn infant, 92
KREMENS, V. (WITH BERGER, S. M., AND BORDEADIS, A. G.), Bronchial adenoma, 417

L

LALL, SEETA, AND KARELITZ, SAMUEL, The treatment of diphtheria and diphtheria carriers with terramycin, 35
LAWSON, WILBUR G., Chronic urinary tract infection in a female child, 263
LEUALLEN, E. E. (WITH PERLMAN, HENRY HARRIS), What the pediatrician should know about the newer ointments and ointment bases, 578
LEVIN, MARCIA (WITH BAUMANN, FRANZ, AND PEARSON, DELBERT E.), Adrenal cortical steroids in management of a case of meningococcemia, 575
LEVY, WALTER E. (WITH MASCIA, ARMOND V., AND CHICK, FORRIS E.), Effects of rubeola on tuberculosis, 294
LEWIS, H. KENT (WITH WATTENBERG, CARL A., ABRAMS, MORRIS, AND LEWIS, JOHN), Bilateral epididymal tuberculosis in an infant, 443
LEWIS, JOHN (WITH WATTENBERG, CARL A., ABRAMS, MORRIS, AND LEWIS, H. KENT), Bilateral epididymal tuberculosis in an infant, 443
LIDGE, RALPH T., AND CHANDLER, FREMONT A., Charcot-Marie-Tooth disease, 152
LINCOLN, EDITH M. (WITH SIFONTES, JOSÉ E., WILLIAMS, R. D. BROOKE, AND CLEMONS, HELEN), Observations on the effect of induced hyperglycemia on the glucose content of the cerebrospinal fluid in patients with hydrocephalus, 243
LIVINGSTON, SAMUEL, The use of Hibicon in the treatment of epilepsy, 673
— (WITH DOYLE, P. J.), The use of Mysoline in the treatment of epilepsy, 413
— (WITH DOYLE, P. J., AND PEARSON, PAUL H.), Use of Milontin in the treatment of petit mal epilepsy (three per second spike and wave dysrhythmia), 164
— (WITH REICHELDERFER, THOMAS E., AND PEARSON, PAUL H.), Thrombocytopenic purpura occurring in association with Paradione (paramethadione) and Dilantin Sodium (phenytoin sodium) therapy, 43
LONGINO, LUTHER A., AND MEEKER, IRVING A., JR., Primary cardiac tumors in infancy, 724
LUBOW, HENRY (WITH SCHWARTZMAN, JOSEPH, AND ZAONTZ, JOHN B.), Chorea minor: preliminary report on six patients treated with combined ACTH and cortisone, 278

M

MAGEE, M. CATHERINE (WITH ABRAMSON, HAROLD, AND GREENBERG, MORRIS), Poliomyelitis in the newborn infant, 167
MAIER, HERBERT C., AND GOULD, WILBUR J., Agenesis of the lung with vascular compression of the tracheobronchial tree, 38
MALONE, H. JOHN (WITH RATNER, BRET, UNTRACHT, SAMUEL, AND RETSINA, MARY), Allergenicity of modified and processed foodstuffs. IV. Orange: allergenicity of orange studied in man, 421
MANSON, GORDON, Acute respiratory disease with special reference to pathogenesis, classification, and diagnosis, 599
MARCUSE, PETER M., BURGER, ROBERT A., AND SALMON, GEORGE W., Hamartoma of the hypothalamus, 301
MARKS, DENNIS N., SHAPIRO, ROBERT S., AND JOSEPH, LESTER G., Roentgenographic diagnosis of coaretation of the aorta in a two-day-old infant, 453
MASCIA, ARMOND V., CHICK, FORRIS E., AND LEVY, WALTER E., Effects of rubeola on tuberculosis, 294
MCCULLOUGH, A. W., Further examples of endocardial cushion defect in production of a cardiac anomaly complex, 429
MCKEY, ROBERT W. (WITH COHEN, ROBERT, O'CONNOR, ROBERT E., TOWNSEND, THOMAS E., AND WEBB, PATRICIA ANN), Western equine encephalomyelitis, 26
MEEKER, IRVING A., JR., (WITH LONGINO, LUTHER A.), Primary cardiac tumors in infancy, 724
MEGEVAND, RENE P., PAUL, RAPHAEL N., AND PARKER, JOSEPH, Single ventricle with diminutive outlet chamber associated with coaretation of the aorta and other cardiac anomalies, 687
MELIN, KARL-AXEL, Pertussis immunization in children with convulsive disorders, 652
MIDDELKAMP, J. N., Varicella in newborn twins, 573
MOHROHISKY, JOHN F. (WITH KEEFER, GEORGE P.), Lead poisoning: roentgenograms of the chest as an aid in diagnosis, 146
MONTGOMERY, THEODORE A. (WITH CARSON, MERL J.), Congenital hyperhidrosis: therapy with Hydergine and Banthine, 274
MOORE, S. (WITH GEDDES, A. K.), Acute (infantile) Gaucher's disease, 61
MYERS, B. (WITH ROTHSCHILD, H. B., AND STORCK, A.), Mesenteric occlusion in a newborn infant, 569

N

NEWMAN, BENJAMIN (WITH ODESSKY, LOUIS, BEDO, ANDREW V., AND WEISLER, HARRY), Encephalitis in children with measles who were given attenuating doses of gamma globulin, 404

— (WITH ODESSKY, LOUIS, BEDO, ANDREW V., JENNINGS, KENNETH G., SANDS, IRVING J., ROSENBLATT, PHILIP, AND WEISLER, HARRY), Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. I. A clinical study of forty-one cases with follow-up studies, 536

— (WITH ODESSKY, LOUIS, BEDO, ANDREW V., ROSENBLATT, PHILIP, JENNINGS, KENNETH G., SANDS, IRVING J., AND WEISLER, HARRY), Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. II. Cerebrospinal fluid and serum studies in measles encephalitis and encephalomyelitis with special reference to phosphorus, cholesterol, and magnesium, 695

O

OBERKIRCHER, OSCAR, J., STAUBITZ, WILLIAM J., AND PARMENTER, FRED J., A clinical study of neuroblastoma, 177

O'CONNOR, ROBERT E. (WITH COHEN, ROBERT, TOWNSEND, THOMAS E., WEBB, PATRICIA ANN, AND MCKEY, ROBERT W.), Western equine encephalomyelitis, 26

ODESSKY, LOUIS, BEDO, ANDREW V., JENNINGS, KENNETH G., SANDS, IRVING J., ROSENBLATT, PHILIP, WEISLER, HARRY, AND NEWMAN, BENJAMIN, Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. I. A clinical study of forty-one cases with follow-up studies, 536

—, —, ROSENBLATT, PHILIP, JENNINGS, KENNETH G., SANDS, IRVING J., WEISLER, HARRY, AND NEWMAN, BENJAMIN, Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. II. Cerebrospinal fluid and serum studies in measles encephalitis and encephalomyelitis with special reference to phosphorus, cholesterol, and magnesium, 695

—, —, WEISLER, HARRY, AND NEWMAN, BENJAMIN, Encephalitis in children with measles who were given attenuating doses of gamma globulin, 404

OKUMOTO, YOSHIO (WITH GREULICH, WILLIAM WALTER, CRISMON, CATHRINE S., AND TURNER, MARGARET L.), The physical growth and development of children who survived the atomic bombing of Hiroshima or Nagasaki, 121

OWNBY, RALPH, JR. (WITH KENDIG, EDWIN L., TREVATHAN, G. EARL, AND SUTTON, LEE E., JR.), The treatment of tuberculous meningitis in children, 532

P

PAPADATOS, C. (WITH KLEIN, R., AND FORTUNATO, J.), Serum corticoids in children under stress, 385

PARKER, GEORGE F. (WITH FREEMAN, MAX E.), Treatment of staphylococcic pericarditis with bacitracin, 720

PARKER, JOSEPH (WITH MEGEVAND, RENE P., AND PAUL, RAPHAEL N.), Single ventricle with diminutive outlet chamber associated with coarctation of the aorta and other cardiac anomalies, 687

PARMENTER, FRED J. (WITH OBERKIRCHER, OSCAR J., AND STAUBITZ, WILLIAM J.), A clinical study of neuroblastoma, 177

PAUL, RAPHAEL N. (WITH MEGEVAND, RENE P., AND PARKER, JOSEPH), Single ventricle with diminutive outlet chamber associated with coarctation of the aorta and other cardiac anomalies, 687

PEARSON, DELBERT E. (WITH BAUMANN, FRANZ, AND LEVIN, MARCIA), Adrenal cortical steroids in management of a case of meningococcemia, 575

PEARSON, PAUL H. (WITH DOYLE, P. J., AND LIVINGSTON, SAMUEL), Use of Milontin in the treatment of petit mal epilepsy (three per second spike and wave dysrhythmia), 164

— (WITH REICHELDERFER, THOMAS E., AND LIVINGSTON, SAMUEL), Thrombocytopenic purpura occurring in association with Paradione (paramethadione) and Dilantin Sodium (phenytoin sodium) therapy, 43

PERLMAN, HENRY HARRIS, AND LEUALLEN, E. E., What the pediatrician should know about the newer ointments and ointment bases, 578

PERSKY, LESTER (WITH HUFFMAN, WILLIAM L., AND JOELSON, JAMES J.), Extravesical ureteral ectopia, 407

PRINTZ, JOSEPH H. (WITH BORENSTINE, JOSEPH), Meconium ileus (complete obstruction) in a premature infant, 208

R

RATNER, BRET, UNTRACHT, SAMUEL, MALONE, H. JOHN, AND RETSINA, MARY, Alllergenicity of modified and processed foodstuffs. IV. Orange: Allergenicity of orange studied in man, 421

REGNIER, EDME, A current summary of strabismus in children, 732

REICHELDERFER, THOMAS E., PEARSON, PAUL H., AND LIVINGSTON, SAMUEL, Thrombocytopenic purpura occurring in association with Paradione (paramethodione) and Dilantin Sodium (phenytoin sodium) therapy, 43

RETSINA, MARY (WITH RATNER, BRET, UNTRACHT, SAMUEL, AND MALONE, H. JOHN), Allergenicity of modified and processed foodstuffs. IV. Orange: allergenicity of orange studied in man, 421

ROSE, WILLIAM (WITH KNAUS, WILLIAM E., AND CAMPOS, JOSEPH), Meigs' syndrome, 88

ROSENBLATT, PHILIP (WITH ODESSKY, LOUIS, BEDO, ANDREW V., JENNINGS, KENNETH G., SANDS, IRVING J., WEISLER, HARRY, AND NEWMAN, BENJAMIN), Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. I. A clinical study of forty-one cases with follow-up studies, 536

— (WITH ODESSKY, LOUIS, BEDO, ANDREW V., JENNINGS, KENNETH G., SANDS, IRVING J., WEISLER, HARRY, AND NEWMAN, BENJAMIN), Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. II. Cerebrospinal fluid and serum studies in measles encephalitis and encephalomyelitis with special reference to phosphorus, cholesterol, and magnesium, 695

— (WITH SMITH, EMIL, AND BEDO, ANDREW V.), Sickle-cell anemia crisis, 655

— (WITH SMITH, EMIL, AND HARRIS, IRWIN L.), Acute poliomyelitis: clinical and statistical study of 263 cases, 9

ROSS, LUCILLE J., A summer camp for children with heart disease, 67

ROTHSCHILD, H. B., STORCK, A., AND MYERS, B., Mesenteric occlusion in a newborn infant, 569

S

SALMON, GEORGE W. (WITH DAESCHNER, C. WM., AND HEYS, FLORENCE M.), Cat-scratch fever, 371

— (WITH MARCUSE, PETER M., AND BURGER, ROBERT A.), Hamartoma of the hypothalamus, 301

SANDS, IRVING J. (WITH ODESSKY, LOUIS, BEDO, ANDREW V., JENNINGS, KENNETH G., ROSENBLATT, PHILIP, WEISLER, HARRY, AND NEWMAN, BENJAMIN), Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. I. A clinical study of forty-one cases with follow-up studies, 536

— (WITH ODESSKY, LOUIS, BEDO, ANDREW V., ROSENBLATT, PHILIP, JENNINGS, KENNETH G., WEISLER, HARRY, AND NEWMAN, BENJAMIN), Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. II. Cerebrospinal fluid and serum studies in measles encephalitis and encephalomyelitis with special reference to phosphorus, cholesterol, and magnesium, 695

SANKEY, H. H. (WITH DONALDSON, J. S., GIRDANY, B. R., AND DONALDSON, W. F.), Osteochondroma of the distal femoral epiphysis, 212

SCHAAR, FRANCES E., Bone marrow aspiration sites in infants and children, 297

SCHWARTZMAN, JOSEPH, ZAONTZ, JOHN B., AND LUBOW, HENRY, Chorea minor: preliminary report on six patients treated with combined ACTH and cortisone, 278

SHAPIRO, ROBERT S. (WITH MARKS, DENNIS N., AND JOSEPH, LESTER G.), Roentgenographic diagnosis of coartation of the aorta in a two-day-old infant, 453

SHINEFIELD, HENRY R., AND TOWNSEND, THOMAS E., Transplacental transmission of Western equine encephalomyelitis, 21

SIFONTES, JOSÉ E., WILLIAMS, R. D. BROOKE, LINCOLN, EDITH M., AND CLEMONS, HELEN, Observations on the effect of induced hyperglycemia on the glucose content of the cerebrospinal fluid in patients with hydrocephalus, 243

SMITH, CARL H., Hypoplastic and aplastic anemias of infancy and childhood: with a consideration of the syndrome of nonhemolytic anemia of the newborn, 457

SMITH, EMIL, HARRIS, IRWIN L., AND ROSENBLATT, PHILIP, Acute poliomyelitis: clinical and statistical study of 263 cases, 9

—, ROSENBLATT, PHILIP, AND BEDO, ANDREW V., Sickle-cell anemia crisis, 655

STADLER, HAROLD E., Neonatal symmetrical pedal gangrene with complete recession, 447

STAUBITZ, WILLIAM J. (WITH OBERKIRCHER, OSCAR J., AND PARMENTER, FRED J.), A clinical study of neuroblastoma, 177

STEVENS, RICHARD C. (WITH TOMSYKOSKI, A. J.), Embryonal cell carcinoma of the liver in infancy, 309

STORCK, A. (WITH ROTHSCHILD, H. B., AND MYERS, B.), Mesenteric occlusion in a newborn infant, 569

SUTTON, LEE E., JR. (WITH HENNIGAR, GORDON R., THABET, RAYMOND, AND BUNDY, WALTER E.), Salmonellosis complicated by pancarditis, 524

— (WITH KENDIG, EDWIN L., JR., OWNBY, RALPH, JR., AND TREVATHAN, G. EARL), The treatment of tuberculous meningitis in children, 532

SZYPULSKI, JOHN T. (WITH HSU, HAN-KUANG), Some problems in the diagnosis and treatment of pulmonary tuberculosis in childhood, 661

T

TALBOT, BEATRICE (WITH BRAZELTON, T. BERRY, AND HOLDER, RICHMOND), Emotional aspects of rheumatic fever in children, 339

THABET, RAYMOND (WITH HENNIGAR, GORDON R., BUNDY, WALTER E., AND SUTTON, LEE E., JR.), Salmonellosis complicated by pancarditis, 524

TOMSYKOSKI, A. J., AND STEVENS, RICHARD C., Embryonal cell carcinoma of the liver in infancy, 309

TOWNSEND, THOMAS E. (WITH COHEN, ROBERT, O'CONNOR, ROBERT E., WEBB, PATRICIA ANN, AND MCKEY, ROBERT W.), Western equine encephalomyelitis, 26

— (WITH SHINEFIELD, HENRY R.), Transplacental transmission of Western equine encephalomyelitis, 21

TRAVATHAN, G. EARL (WITH KENDIG, EDWIN L., JR., OWNBY, RALPH, JR., AND SUTTON, LEE E., JR.), The treatment of tuberculous meningitis in children, 532

TURIN, RICHARD, BLOCH, HARRY, AND KAZNETZ, JOSEPH, Hematemesis: report of a case in an infant due to arterial bleeding from the base of the tongue, 96

TURNER, MARGARET L. (WITH GREULICH, WILLIAM WALTER, AND CRISMON, CATHRINE S.), The physical growth and development of children who survived the atomic bombing of Hiroshima or Nagasaki, 121

U

UNTRACHT, SAMUEL (WITH RATNER, BRET, MALONE, H. JOHN, AND RETSINA, MARY), Allergenicity of modified and processed foodstuffs. IV. Orange: allergenicity of orange studied in man, 421

W

WARD, ROBERT, Recent progress in poliomyelitis research, 98

WARNER, MARY P., AND GROVE, FRANCES E., A study of forty cases of ringworm of the scalp, 267

WATTENBERG, CARL A., ABRAMS, MORRIS, LEWIS, H. KENT, AND LEWIS, JOHN, Bilateral epididymal tuberculosis in an infant, 443

WEBB, PATRICIA ANN (WITH COHEN, ROBERT, O'CONNOR, ROBERT E., TOWNSEND, THOMAS E., AND MCKEY, ROBERT W.), Western equine encephalomyelitis, 26

WEISLER, HARRY (WITH ODESSKY, LOUIS, BEDO, ANDREW V., AND NEWMAN, BENJAMIN), Encephalitis in children with measles who were given attenuating doses of gamma globulin, 404

— (WITH ODESSKY, LOUIS, BEDO, ANDREW V., JENNINGS, KENNETH G., SANDS, IRVING J., ROSENBLATT, PHILIP, AND NEWMAN, BENJAMIN), Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. I. A clinical study of forty-one cases with follow-up studies, 536

— (WITH ODESSKY, LOUIS, BEDO, ANDREW V., ROSENBLATT, PHILIP, JENNINGS, KENNETH G., SANDS, IRVING J., AND NEWMAN, BENJAMIN), Therapeutic doses of gamma globulin in the treatment of measles encephalitis and encephalomyelitis. II. Cerebrospinal fluid and serum studies in measles encephalitis and encephalomyelitis with special reference to phosphorus, cholesterol, and magnesium, 695

WILLIAMS, D. BROOKE (WITH DUCEY, JAMES), Transcutaneous absorption of boric acid, 644

WILLIAMS, R. D., BROOKE (WITH SIFONTES, JOSÉ E., LINCOLN, EDITH M., AND CLEMONS, HELEN), Observations on the effect of induced hyperglycemia on the glucose content of the cerebrospinal fluid in patients with hydrocephalus, 243

WOLFISH, MARTIN G., Acute gastroenteritis. A review of 518 cases treated at the Hospital for Sick Children during 1951 and 1952, 675

Z

ZAONTZ, JOHN B. (WITH SCHWARTZMAN, JOSEPH, AND LUBOW, HENRY), Chorea minor: preliminary report on six patients treated with combined ACTH and cortisone, 278

SUBJECT INDEX*

A

Accidental poisoning, death from (editorial), 368
in children in Scotland, reported increase in (editorial), 750
ACTH and cortisone combined, therapy for chorea minor (Schwartzman et al.), 278
Adrenal cortical steroids in management of case of meningococcemia (Baumann et al.), 575
Agenesis of lung with vascular compression of tracheobronchial tree (Maier and Gould), 38
Allergy to orange studied in man: allergenicity of modified and processed foodstuffs (Ratner et al.), 421
American Pediatric Society, founder members of (editorial), 235
Samoa, leukemia in (Earle), 398
Anemia, sickle-cell, with unusual bone changes (Ivy and Howard), 312
Anemias, hypoplastic and aplastic, of infancy and childhood: with consideration of syndrome of nonhemolytic anemia of newborn (Smith), 457
Antibiotics, frivolous use of (editorial), 234
Apology to Texas (editorial), 120
Athletic competition for children (editorial), 118
Atomic bombing of Hiroshima or Nagasaki, physical growth and development of children who survived (Greulich et al.), 121

B

Bacitracin and Sulfasuxidine, treatment of pinworm (Enterobius vermicularis) infection with (Chan and Brown), 290
treatment of staphylococcic pericarditis with (Freeman and Parker), 720
Banthine and Hydergine, therapy for congenital hyperhidrosis (Carson and Montgomery), 274
Bone marrow aspiration sites in infants and children (Schaar), 297
Book Reviews:
ABC für Zuckerkranke (Bertram), 494
Advances in pediatrics (Levine, ed.), 625
American pediatric directory (Smith), 625
Blum-Fieldsteel development charts, 365
Bright children (Cutts and Moseley), 492
Book Reviews—Cont'd
Child development (Martin and Stender), 492
Clinical orthopaedics (DePalma, ed.), 365
Congenital anomalies of the heart and great vessels (Schnitker), 365
Developmental psychology (Hurlock), 745
Food for life (Gerard, ed.), 117
Handbook of orthopaedic surgery (Shands, Jr.), 625
Health services for the child (Schlesinger), 745
Kinderärztliche Notfallfibel (de Rudder), 233
Les Antibiotiques et les Maladies de l'Enfance, 494
Modern concepts of communicable disease (Greenberg and Matz), 364
Nervous system (Netter), 233
Nutritional studies in adolescent girls and their relation to tuberculosis (Johnston), 625
Reducing emotional trauma in hospitalized children (Albany Medical College), 365
Rehabilitation of the physically handicapped (Kessler), 233
Sexual behavior in the human female (Kinsey et al.), 624
Speech problems of school children, 745
Teach your child to read (Cox), 117
The book of health (Clark, Jr., and Cumley, eds.), 493
The conception of disease (Riese), 233
The surgery of infancy and childhood (Gross), 492
Boric acid poisoning (Ducey and Williams), 644; (editorial), 746; (Goldbloom and Goldbloom), 631
Bronchial adenoma (Berger et al.), 417

C

Carcinoma, embryonal cell, of liver in infancy (Tomsykoski and Stevens), 309
Cardiac disease, relation of, to feelings of fear (Freed and Cruickshank), 483
tumors, primary, in infancy (Longino and Meeker), 724
Cat-scratch fever (Daeschner et al.), 371
Central nervous system disease in newborn infant, unusual form of (Berrey and Dannenbring), 388
infections of, clinic on (Children's Hospital, San Francisco), 316

*July, pp. 1-120; August, pp. 121-236; September, pp. 237-370; October, pp. 371-498; November, pp. 499-630; December, pp. 631-750.

Cerebrospinal fluid in patients with hydrocephalus, effect of induced hyperglycemia on glucose content of, observations on (Sifontes et al.), 243
Charcot-Marie-Tooth disease (Lidge and Chandler), 152
Chorea minor: preliminary report on six patients treated with combined ACTH and cortisone (Schwartzman et al.), 278
Clinical Conferences:
Communicable Disease Department, Children's Hospital, San Francisco, Calif., clinic on infections of central nervous system, 316
St. Louis Children's Hospital, clinic on case of tetanus, 220
Coaretation of aorta in two-day-old infant, roentgenographic diagnosis of (Marks et al.), 453
surgically acquired hypothyroidism in child with (Johnson and Bruton), 449
Comments on Current Literature:
histoplasmosis, 489
infantile eczema, 359
infection in newborn baby, 112
poliomyelitis: incidence of subclinical infection during epidemic, 620
smallpox vaccination and pregnancy, 229
spread of rickettsialpox, 740
Congenital defects, rehabilitation of children with numerous (Kanof and Bernstein), 434
heart disease:
roentgenographic diagnosis of coaretation of aorta in two-day-old infant (Marks et al.), 453
single ventricle with diminutive outlet chamber associated with coaretation of aorta and other cardiac anomalies (Megevand et al.), 687
surgically acquired hypothyroidism in child with coaretation of aorta (Johnson and Bruton), 449
Convulsive disorders, children with, immunize against pertussis? (editorial), 746
pertussis immunization in (Melin), 652
Cortisone and ACTH combined, therapy for chorea minor (Schwartzman et al.), 278
Current pediatric references (editorial), 370
Cytomegalic inclusion disease, generalized (Bacala and Burke), 712

D

Dental caries among elementary school children in Oregon (Hadjimarkos), 393
Diphtheria and diphtheria carriers, treatment of, with terramycin (Lall and Karelitz), 35

E

Editor's Column:
apology to Texas, 120
athletic competition for children, 118
Editor's Column—Cont'd
boric acid, 746
care and cost of case of tetanus, 236
current pediatric references, 370
death from accidental poisoning, 368
distribution of polio in United States, 366
founder members of American Pediatric Society, 235
frivolous use of antibiotics, 234
gifts—with strings attached, 495
over the editor's desk, 626
reported increase in accidental poisoning in children in Scotland, 750
should child with convulsive disorders be immunized against pertussis? 746
surgery in children's hospital, 497
Encephalitis in children with measles who were given attenuating doses of gamma globulin (Odessky et al.), 404
measles, and encephalomyelitis, therapeutic doses of gamma globulin in treatment of (Odessky et al.), 536, 695
transplacental transmission of Western equine encephalomyelitis (Shinefield and Townsend), 21
Western equine encephalomyelitis (Cohen et al.), 26
Endocardial cushion defect in production of cardiac anomaly complex, further examples of (McCullough), 429
Enterobiasis (oxyuriasis), treatment of; need for special sleeping garment (Kean), 47
Enterobius vermicularis, treatment of pinworm infection with bacitracin and Sulfasuxidine (Chan and Brown), 290
Epididymal tuberculosis, bilateral, in infants (Wattenberg et al.), 443
Epilepsy, use of Hibicon in treatment of (Livingston), 673
of Mysoline in treatment of (Doyle and Livingston), 413

F

Fear, feelings of, relation of cardiac disease to (Freed and Cruickshank), 483
Feeding, infant, brief history of (Davidson), 74

G

Galactose, blood, in infants and children (Hartmann et al.), 1
Gamma globulin, children with measles who were given attenuating doses of, encephalitis in (Odessky et al.), 404
dosage and measles control (Bivings), 401
therapeutic doses of, in treatment of measles encephalitis and encephalomyelitis, cerebrospinal fluid and serum studies in, with

Gamma globulin—Cont'd
special reference to phosphorus, cholesterol, and magnesium (Odessky et al.), 695
clinical study of forty-one cases with follow-up studies (Odessky et al.), 536
Gangrene, pedal, symmetrical, neonatal, with complete recession (Stadler), 447
Gastroenteritis, acute (Wolfish), 675
Gaucher's disease, acute (infantile) (Geddes and Moore), 61
Gifts—with strings attached (editorial), 495

H

Hamartoma of hypothalamus (Marcuse et al.), 301
Heart disease, summer camp for children with (Ross), 67
Hematemesis (Turin et al.), 96
Hibicon, use of, in treatment of epilepsy (Livingston), 673
Histoplasmosis (Blattner), 489
Homosexual behavior in children (Bakwin and Bakwin), 108
Hydergine and Banthine, therapy for congenital hyperhidrosis (Carson and Montgomery), 274
Hydrocephalus, patients with, observations on effect of induced hyperglycemia on glucose content of cerebrospinal fluid in (Sifontes), 243
Hyperglycemia, induced, effect of, on glucose content of cerebrospinal fluid in patients with hydrocephalus, observations on (Sifontes et al.), 243
Hyperhidrosis, congenital, therapy with Hydergine and Banthine (Carson and Montgomery), 274
Hypoglycemia, neonatal, observations related to (Desmond), 253
Hypoplastic and aplastic anemias of infancy and childhood: with consideration of syndrome of nonhemolytic anemia of newborn (Smith), 457
Hypothalamus, hamartoma of (Marcuse et al.), 301
Hypothyroidism, surgically acquired, in child with coarctation of aorta (Johnson and Bruton), 449

I

Infancy and childhood, hypoplastic and aplastic anemias of: with consideration of syndrome of nonhemolytic anemia of newborn (Smith), 457
embryonal cell carcinoma of liver in (Tomsykoski and Stevens), 309
primary cardiac tumors in (Longino and Meeker), 724
Infant, bilateral epididymal tuberculosis in (Wattenberg), 443
feeding, brief history of (Davidson), 74
Infant—Cont'd
two-day-old, coarctation of aorta in, roentgenographic diagnosis of (Marks et al.), 453
Infantile eczema (Blattner), 359
Infants and children, blood galactose in (Hartmann et al.), 1
bone marrow aspiration sites in (Schaar), 297
Infection in newborn baby (Blattner), 112
Infusion, scalp-vein, pediatric, Gardner-Murphy, outfit, use of (Abella), 709

L

Lead poisoning: roentgenograms of chest as aid in diagnosis (Keefer and Mokrohisky), 146
Leukemia in American Samoa (Earle), 398
Liver, embryonal cell carcinoma of, in infancy (Tomsykoski and Stevens), 309

M

Measles, children with, who were given attenuating doses of gamma globulin, encephalitis in (Odessky et al.), 404
control, gamma globulin dosage and (Bivings), 401
encephalitis and encephalomyelitis, therapeutic doses of gamma globulin in treatment of (Odessky et al.), 536, 695
Meconium ileus (complete obstruction) in premature infant (Borenstine and Printz), 208
Medical Progress:
acute respiratory disease with special reference to pathogenesis, classification, and diagnosis (Manson), 599
current summary of strabismus in children (Regnier), 732
hypoplastic and aplastic anemias of infancy and childhood: with consideration of syndrome of nonhemolytic anemia of newborn (Smith), 457
recent progress in poliomyelitis research (Ward), 98
Meigs' syndrome (Knaus et al.), 88
Meningitis in children, three unusual cases of (Chigier), 54
Meningococcemia, adrenal cortical steroids in management of case of (Baumann et al.), 575
Mesenteric occlusion in newborn infant (Rothschild et al.), 569
Milontin, use of, in treatment of petit mal epilepsy (three per second spike and wave dysrhythmia) (Doyle et al.), 164
Mysoline, use of, in treatment of epilepsy (Doyle and Livingston), 413

N

Neuroblastoma, clinical study of (Oberkircher et al.), 177
Newborn infant, infection in (Blattner), 112
mesenteric occlusion in (Rothschild et al.), 569
paroxysmal auricular tachycardia associated with Wolff-Parkinson-White syndrome in (Kreidberg and Dushan), 92
poliomyelitis in (Abramson et al.), 167
unusual form of central nervous system disease in (Berrey and Dannenbring), 388
twins, varicella in (Middelkamp), 573
News and Notes, 114, 232, 362, 492, 623, 743

O

Ointments and ointment bases, newer, what pediatrician should know about (Perlman and Leuallen), 578
Orange, allergenicity of, studied in man: allergenicity of modified and processed foodstuffs (Ratner et al.), 421
Osteochondroma of distal femoral epiphysis Donaldson et al.), 212

P

Pancarditis, salmonellosis complicated by (Hennigar et al.), 524
Paroxysmal auricular tachycardia associated with Wolff-Parkinson-White syndrome in newborn infant (Kreidberg and Dushan), 92
tachycardia, congenital (Anderson and Adams), 668
Pedal gangrene, symmetrical, neonatal, with complete recession, 447
Pediatric Therapeutics:
what pediatrician should know about newer ointments and ointment bases (Perlman and Leuallen), 578
Penicillin injections, effect of, during acute poliomyelitis (Brown), 174
Pericarditis, staphylococcic, treatment of, with bacitracin (Freeman and Parker), 720
Pertussis immunization in children with convulsive disorders (Melin), 652
should child with convulsive disorders be immunized against? (editorial), 746
Petit mal epilepsy (three per second spike and wave dysrhythmia), use of Milontin in treatment of (Doyle et al.), 164
Physical growth and development of children who survived atomic bombing of Hiroshima or Nagasaki (Greulich et al.), 121
Poisoning, accidental, death from (editorial), 368
in children in Scotland, reported increase in (editorial), 750
Poisoning—Cont'd
boric acid (Ducey and Williams), 644; (editorial), 746; (Goldbloom and Goldbloom), 631
lead: roentgenograms of chest as aid in diagnosis (Keefer and Mokrohisky), 146
Poliomyelitis, acute (Smith et al.), 9
effect of penicillin injections during (Brown), 174
distribution of, in United States (editorial), 366
in newborn infant (Abramson et al.), 167
incidence of subclinical infection during epidemic (Blattner), 620
research, recent progress in (Ward), 98
Post hoc, ergo propter hoc (Clifford), 237
Pregnancy, smallpox vaccination and (Blattner), 229
Premature infant, developing fundus oculi of, and its relationship to retrolental fibroplasia (Fletcher), 499
meconium ileus (complete obstruction) in (Borenstine and Printz), 208
Priscoline, sickle-cell anemia crisis treated with (Smith et al.), 655
Psychologic Aspects of Pediatrics:
early development of children with schizophrenia (Bakwin), 217
emotional aspects of rheumatic fever in children (Brazelton et al.), 339
homosexual behavior in children (Bakwin and Bakwin), 108
relation of cardiac disease to feelings of fear (Freed and Cruickshank), 483
Pulmonary tuberculosis in childhood, some problems in diagnosis and treatment of (Hsu and Szypulski), 661

R

Rehabilitation of child with numerous congenital defects (Kanof and Bernstein), 434
Respiratory disease, acute, with special reference to pathogenesis, classification, and diagnosis (Manson), 599
Retrolental fibroplasia, developing fundus oculi of premature infant and its relationship to (Fletcher), 499
Rheumatic fever in children, emotional aspects of (Brazelton et al.), 339
Rickettsialpox, spread of (Blattner), 740
Ringworm of scalp, study of forty cases of (Warner and Grove), 267
Rubeola, effects of, on tuberculosis (Mascia et al.), 294

S

Salmonellosis complicated by pancarditis (Hennigar et al.), 524
Scalp-vein infusion outfit, pediatric, Gardner-Murphy, use of (Abella), 709
Schizophrenia, early development of children with (Bakwin), 217

Serum corticoids in children under stress (Klein et al.), 385
Sickle-cell anemia crisis (Smith et al.), 655
with unusual bone changes (Ivy and Howard), 312
Smallpox vaccination and pregnancy (Blattner), 229
Spinal cord tumors in children (Anderson and Carson), 190
Strabismus in children, current summary of (Regnier), 732
Stress, children under, serum corticoids in (Klein et al.), 385
Sulfasuxidine and bacitracin, treatment of pinworm (Enterobius vermicularis) infection with (Chan and Brown), 290
Summer camp for children with heart disease (Ross), 67
Surgery in children's hospital (editorial), 497

T

Terramycin, treatment of diphtheria and diphtheria carriers with (Lall and Karelitz), 35
Tetanus, case of, care and cost of (editorial), 236
clinic on (St. Louis Children's Hospital), 220
Thrombocytopenic purpura occurring in association with Paradione (paramethadione) and Dilantin Sodium (phenytoin sodium) therapy (Reichelderfer et al.), 43
Tuberculosis, effects of rubeola on (Mascia et al.), 294
Tuberculous meningitis in children, treatment of (Kendig et al.), 532
Tumors, cardiac, primary, in infancy (Longino and Meeker), 724

U

Ureteral ectopia, extravesical (Huffman et al.), 407
Urinary tract infection, chronic, in female child (Lawson), 263

V

Vaccination, smallpox, and pregnancy (Blattner), 229
Varicella in newborn twins (Middelkamp), 573

W

Western equine encephalomyelitis (Cohen et al.), 26
transplacental transmission (Shinefield and Townsend), 21

Current Pediatrics References

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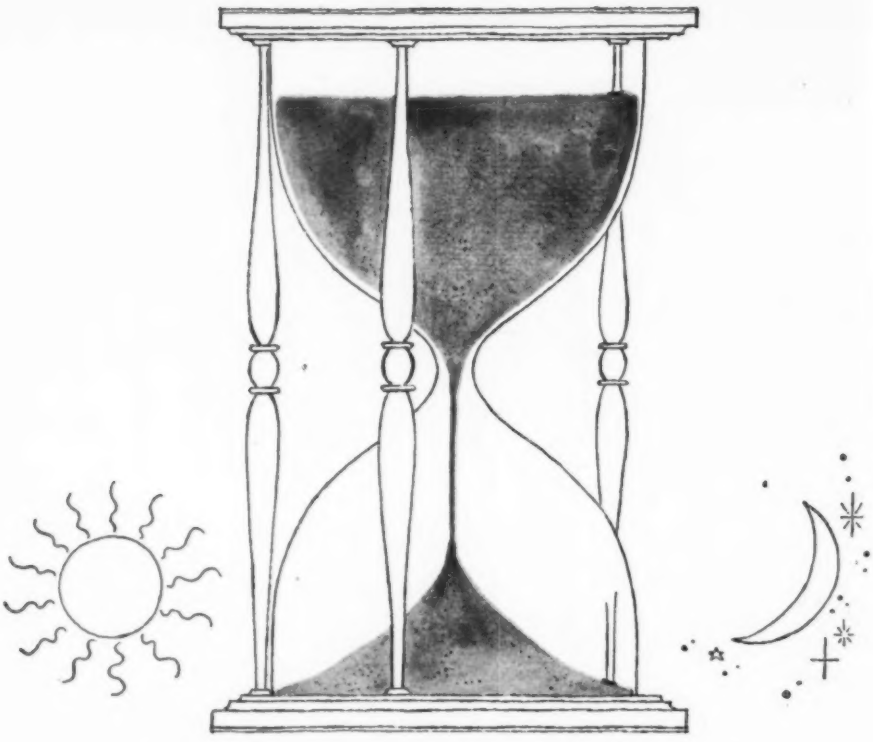

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Feed to the very young in formula—later by spoon. 7 kinds for variety—all 100% meat. Beef, Lamb, Veal, Pork, Liver, Heart, Liver-and-Bacon.

New—Swift's Strained Egg Yolks for Babies. Special, new process sterilizes yolks for purity—actually safer for babies than fresh eggs. A rich source of iron. Pre-cooked creamy-smooth, ready to serve.

Swift's FOREMOST NAME IN Meats for Babies

NUTRITION IS OUR BUSINESS

Must Baby Bottles Be Sterilized?

Yes . . . but many nursing bottles cannot be kept sterile in the home — because:

1. Only the Hygeia breast-shaped nipples actually prevent baby's mouth from touching the nipple retaining ring, which is not sterile because it must be tightened, after sterilization.

2. All other baby nipples permit baby's mouth to press against this unsterile ring, because only Hygeia has the exclusive breast-shape.

3. The exclusive Hygeia breast-shaped nipple is not only safer for baby, but healthier, too. Because it is so like the shape of mother's breast, it gives baby natural exercise of his facial muscles, and helps to satisfy his normal sucking urge.

Hygeia also offers these other features!

4. **WIDE MOUTH.** Easier to clean and fill.
5. **NO CLOG VENTS.** No holes to clean.
6. **NIPPLES** can easily be turned inside out for thorough cleaning.
7. **TAPERED SHAPE.** Baby gets whole bottle without excessive tipping.
8. **WIDER BASE.** Not easily tipped over.

Hygeia Standard Nursers are also available with "stretch over" nipples.

HYGEIA FINEST BOTTLE MONEY CAN BUY

especially for the carriage trade...

Children like Vi-Penta Drops because they taste good. Mothers like them because they are easy to give in milk, fruit juice, formula or dropped directly on the tongue. Doctors like them because they provide required amounts of vitamins A, C, D, and important B-complex factors, and because they're dated to insure full potency. Vi-Penta® Drops 'Roche' in packages of 15, 30 and 60 cc with calibrated dropper.

RHINALGAN®

NASAL DECONGESTANT

Uniformly *Safe!* FOR INFANTS • CHILDREN ADULTS AND AGED

DOES NOT CONTAIN ANY ANTIBIOTIC

Does not affect

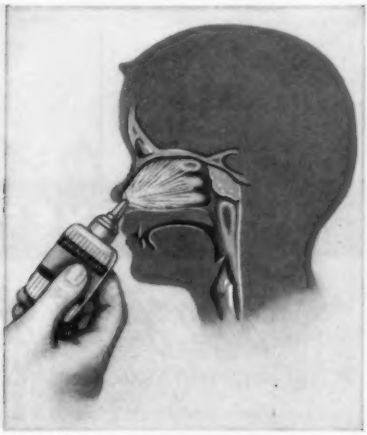
BLOODPRESSURE
RESPIRATION
CENTRAL NERVOUS SYSTEM

ENTIRELY Safe! in

CARDIAC—DIABETIC
PREGNANCY—THYROID
AND HYPERTENSION CASES

Authoritative Proof sent on request.

COMPLETELY FREE OF SIDE-EFFECTS... no cumulative action...no overdosage problem...non-toxic.



For *Safety!* USE RHINALGAN

NOW Modified Formula assures *PLEASANT, PALATABLE TASTE!*

FORMULA: Desoxyephedrine Saccharinate 0.50% w/v in an isotonic aqueous solution with 0.02% Laurylammonium saccharin. Flavored. pH 6.4.

Available on YOUR prescription only!

Reference to RHINALGAN:

1. Van Alyea, O. E., and Donnelly, W. A.: E.E.N.&T. Monthly, 31, Nov. 1952.
2. Fox, S. L.: AMA Arch. Otolaryn., 53, 607-609, 1951.
3. Molomut, N., and Harber, A.: N.Y. Phys., 34, 14-18, 1950.
4. Lett, J. E., (Lt. Col. MC-USAF) Research Report, Dept. Otolaryn., USAF School Aviat. Med., 1952.
5. Hamilton, W. F., and Turnbull, F. M.: J. Amer. Pharm. Ass'n., 7, 378-382, 1950.
6. Browd, Victor L.: Rehabilitation of Hearing, 1950.
7. Kugelmass, I. Newton: Handbook of the Common Acute Infectious Diseases, 1949.

NEW O TOS-MO-SAN—A specific in Suppurative Ear Infections (Acute or Chronic).

AURALGAN—After 40 years STILL the auralgesic and decongestant.

RECTALGAN-Liquid—For symptomatic relief in: Hemorrhoids, Pruritus, Perineal Suturing

DOHO CHEMICAL CORP., 100 Varick Street, New York 13, N. Y.

TWO CORNERSTONES

of infant formulae

MEYENBERG EVAPORATED GOAT MILK has been prescribed by physicians since 1934 as a superior natural milk for patients allergic to Cow's Milk Lactalbumin. By specifying Meyenberg, you make certain of prescription-quality **Grade A goat milk,** sterilized after sealing in special vacuum packed enamel-lined cans. Available in 14 oz. cans at all pharmacies.

HI-PRO: SPECIALLY PREPARED SPRAY DRIED MODIFIED COW'S MILK to provide maximum protein levels without excessive fats or carbohydrates. A flexible basic milk food of exceptional value in the feeding of the infant with fat intolerance, for the premature or for routine feeding. **CONTAINS PROTEIN 41% – FAT 14%.** Available in 1 lb. vacuum tins at all pharmacies.

SEND FOR SAMPLES AND LITERATURE.

PRODUCTS

Jackson-Mitchell Pharmaceuticals, Inc.

• CULVER CITY • CALIFORNIA • SINCE 1934 •

In the UNITED STATES
(4 out of every 5 Bottle Fed Babies)
In CANADA, MEXICO
and Around The World..

Famous *patented* Evenflo
TWIN AIR VALVE NIPPLE
provides colic-free,
precision feeding.

★ *EVENFLO NURSER*
is the overwhelming favorite

- Millions of healthy, happy babies are raised annually with Evenflo.
- Independent surveys by two national magazines show that 4 out of every 5 bottle fed babies in the U. S. A. are using Evenflo.
- Though often imitated, no nurser approaches Evenflo in its colic-free, smooth, precision feeding. Millions of satisfied users!
- No Nurser is handier for mother to use.
- For feeding perfection, utility and topmost value, none equal the constantly improved Evenflo Nurser.

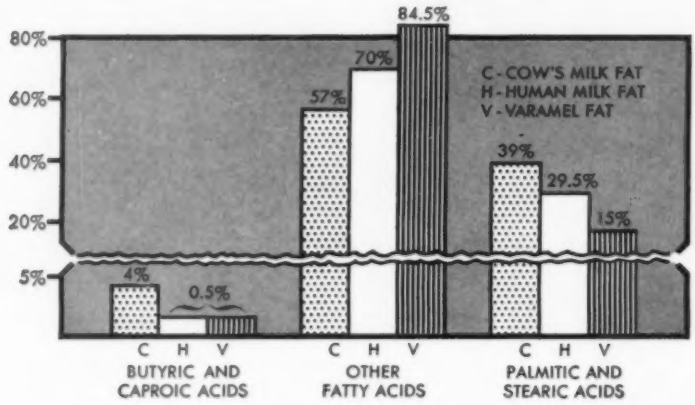
Popular Evenflo Units
4- & 8-oz. sizes **25¢**
Evenflo Colorgrad Units
(ounces in red) **30¢**
Evenflo Deluxe Units
(Pyrex br. glass) **40¢**
Extra Nipples & Parts 10¢
Evenflo 40-pc. Layette Feeding Set $3.00

Evenflo® precision feeding

MADE BY THE PYRAMID RUBBER CO., RAVENNA, OHIO, U.S.A. – "Specialists in Baby Feeding Equipment"

THE *Fats* IN VARAMEL

ARE READILY TOLERATED AND DIGESTED



Varamel provides approximately 85% of its fatty acid composition in the more readily tolerated and digestible range (as shown in the fat chart above). This compares with 57% for cow's milk fat and 70% for the fat of human milk.

The fat composition of Varamel is only one of the reasons why this product is used so successfully in feeding the term newborn infant, the premature infant, and the older infant who does not "handle" butterfat.

Because Varamel is a high-quality* evaporated-type milk containing no added sugar, it may be used in the same manner as any evaporated or bottled milk.

Varamel is ethically promoted and ethically distributed.

THE BAKER LABORATORIES, INC., Cleveland 3, Ohio
Plant: East Troy, Wisconsin • *Division Offices:* Atlanta, Dallas, Denver, Greensboro, N. C., Los Angeles, San Francisco, Seattle

ACCEPTED COUNCIL ON FOODS AND NUTRITION AMERICAN MEDICAL ASSOCIATION

*Made from Grade A milk (U.S. Public Health Service Milk Code) which has been modified by replacement of the milk fat with animal and vegetable oils and by the addition of vitamins and iron. No carbohydrate has been added.

VARAMEL

'POLYSPORIN'

brand

POLYMYXIN B—BACITRACIN OINTMENT

ANTI-GRAM-POSITIVE

BROAD SPECTRUM

ANTI-GRAM-NEGATIVE

Each gram of
'POLYSPORIN' OINTMENT contains:
'AEROSPORIN'® brand Polymyxin B Sulfate 10,000 Units
BACITRACIN 500 Units

BURROUGHS WELLCOME & CO. (U. S. A.) INC.
Tuckahoe 7, N. Y.

INDICATIONS

- infected wounds resulting from trauma or surgery
- infected burns
- infected skin grafts
- abscesses and ulcers in any accessible location
- furuncles
- pyoderma
- ecthyma
- folliculitis
- infectious eczematoid dermatitis
- impetigo
- acne
- styes
- external ear infections
- eye infections such as:
  - conjunctivitis
  - blepharoconjunctivitis
  - scleritis
  - keratitis
  - dacryocystitis, etc.

SECONDARY INFECTIONS
superimposed on any dermatological condition

AVAILABLE IN
tubes of
½ oz.
1 oz.
⅛ oz. ophthalmic

complete information will be sent on request

ANOTHER *first*

FOR DIAPARENE® CHLORIDE

(NO BORIC ACID)

first ANTI-ENZYMATIC SPECIFIC FOR "SORE-BOTTOM"
IN THE NEWBORN AND PERIANAL DERMATITIS FROM DIARRHEA

DIAPARENE PERI-ANAL is the *first* water-repellent to embrace the concept that perianal dermatitis may be caused by stool enzymatic action on the skin . . . by providing anti-enzymatic as well as antibacterial action.

CONTAINS: Di-isobutyl cresoxy ethoxy ethyl di-methyl benzyl ammonium chloride monohydrate, zinc oxide, starch, cod liver oil and casein in a water-repellent base.

SUPPLIED: One ounce tubes and one pound jars

first ANTISEPTIC BABY POWDER PROMOTED AS BORIC ACID-FREE! . . . . . .

first NON-TOXIC, ANTISEPTIC DIAPER RINSE FOR AMMONIA DERMATITIS .

first WATER MISCIBLE OINTMENT FOR URINARY SKIN IRRITATIONS . . . . .

PHARMACEUTICAL DIVISION, HOMEMAKERS' PRODUCTS CORPORATION, 380 SECOND AVENUE, NEW YORK 10, N. Y.—TORONTO 10, CANADA

MUMPS

immune serum

(Human)

Goes to work immediately to prevent mumps and to aid in preventing mumps complications

Administered within first 7 days exposure, serum confers passive immunity for approximately 10 to 14 days. In treatment there is some evidence that the serum prevents serious complications if administered early and in adequate amount. (J. A. M. A. *149*: 1360, Aug. 2, 1952.)

Available 20 cc. irradiated, dried serum with suitable diluent for restoration.

Hyland

HYLAND LABORATORIES 4501 Colorado Blvd., Los Angeles 39, Calif. • 248 S. Broadway, Yonkers 5, N.Y.

Meat...

and the Correction of Negative Nitrogen Balance

Many factors can lead to negative nitrogen balance—surgery, trauma, burns, infection, anorexia, vomiting, and diarrhea. Prompt correction of negative nitrogen balance is imperative because, as long as a nitrogen deficit exists, recovery is retarded, wound healing is slowed, blood regeneration is impeded, antibody production is reduced, weight is lost, and strength is not regained.

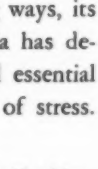
Yet all too frequently patients are permitted to develop—or adequate measures are not taken to correct—a negative nitrogen balance. It has been pointed out that a frequent cause for this complication is failure to recognize the increased protein needs associated with metabolic stress, with resultant failure to adjust the protein content of the diet accordingly.*

Meat in generous amounts can go far in overcoming negative nitrogen balance, regardless of cause. In surgery, the present concept of early postoperative alimentation makes possible prompt adjustment of the diet to provide the biologically complete proteins so generously supplied by meat. Furthermore, since meat can be prepared in an almost endless variety of appetizing ways, its inclusion in the diet aids in reawakening the appetite when anorexia has developed. Meat also supplies significant amounts of B vitamins and essential minerals which are also required in larger amounts during periods of stress.

---

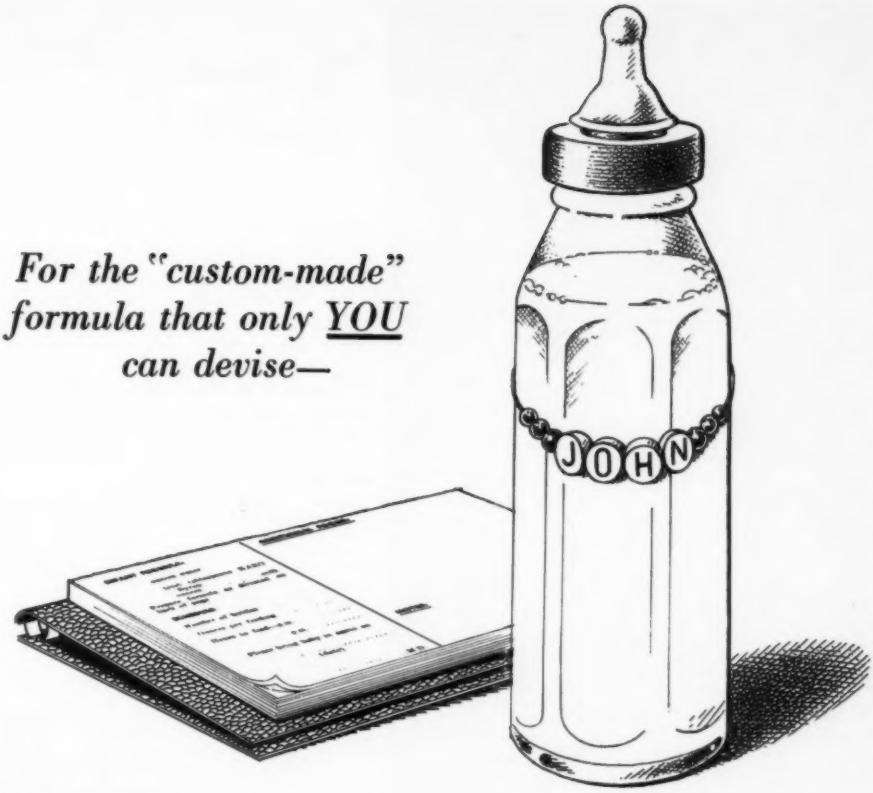
*Pollack, H., and Halpern, S. L., in collaboration with the Committee on Therapeutic Nutrition, Food and Nutrition Board: Therapeutic Nutrition, Publication 234, National Academy of Sciences—National Research Council, 1952.

The Seal of Acceptance denotes that the nutritional statements regarding meat made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.



American Meat Institute
Main Office, Chicago...Members Throughout the United States

For the "custom-made" formula that only YOU can devise—



KARO Syrup . . . the ideal milk modifier for balanced caloric distribution.

Since no two babies will ever be alike —there will always be a need for "custom-made" formulas—the kind only a doctor can prescribe.

The average such formula should, as you know, yield 60% or more of its total calories in carbohydrate form. However, since cow's milk yields only about 28% of its calories in this form—Karo Syrup helps make up the difference.

The Big Advantages of KARO

1. A balanced mixture of dextrins, maltose and dextrose—that may be gradually absorbed, easily digested. Well tolerated, hypoallergenic. Reduces the possibility of fermentation and irritation.
2. Sterilized, bacteria-free—safe for feeding prematures, newborns, other infants.
3. Easy for mother to use, because it's a liquid (soluble in hot or cold fluids). Economical and readily available.
4. Red Label and Blue Label Karo are interchangeable in all formulas (one tablespoon of either yields 60 calories).

Medical Division

CORN PRODUCTS REFINING COMPANY • 17 Battery Place, New York 4, New York

"To Your Patient's Health!"

Good nutrition and good health go hand in hand! Knox Concentrated Gelatine Drink is a worthy protein dietary adjuvant where high protein diet is indicated.

First used by the profession in this form about fifteen years ago, increasing clinical usage demonstrates its professional acceptance. Up to 60 grams of Knox Gelatine in the concentrated drink have been administered daily with no allergic reactions.[1] It contains 25 per cent glycine and 7 out of 8 essential amino acids, as well as 9 other accepted aminos. Knox Gelatine is low in sodium, has a pH of 6.2-6.4, is pure protein with no sugar and no flavoring.

1. Reich, C., and Mulinos, M. G., Treatment of Refractory Nutritional Anemia with Gelatine. Bull. N. Y. Med. Coll. March 1953.

How to Administer Knox Gelatine Concentrate Drink!

Each envelope of Knox Gelatine contains 7 grams which the patient is directed to pour into a ¾ glass of orange juice, other fruit juices, or water, not iced. Let the liquid absorb the gelatine, stir briskly, and drink at once. If it thickens, add more liquid and stir again. Two envelopes or more a day are average minimal doses. Each envelope contains but 28 calories.

For Your Patient's Protection!

Be sure you specify KNOX so that your patient does not mistakenly get factory-flavored gelatine dessert powders which are 85% sugar.

You are invited to send for brochures on diets of Diabetes, Colitis, Peptic Ulcer... Low Salt, Reducing, Liquid, and Soft Diets. KNOX GELATINE, JOHNSTOWN, N.Y. Dept. JP.

Available at grocery stores in 4-envelope family size and 32-envelope economy size packages.

KNOX GELATINE U.S.P.
ALL PROTEIN • NO SUGAR

when nausea and vomiting bring a plea for help . . .

suggest first aid with . . .

EMETROL®

PHOSPHORATED CARBOHYDRATE SOLUTION

a safe, pleasant-tasting, oral antiemetic . . .

effective in 6 out of 7 cases of functional vomiting[1] . . . reduces gastrointestinal smooth muscle contractions physiologically . . . contains no antihistaminics, barbiturates, or other drugs . . . also useful in nausea of pregnancy, and for drug- or anesthetic-induced vomiting

1. Bradley, J. E., et al.: J. Pediat. 38:41, 1951; Idem: Amer. Acad. Pediat., meeting Oct. 16, 1951.

IMPORTANT: EMETROL is stabilized at an optimal physiologic pH level. Dilution would upset this careful balance. For this reason, EMETROL is always taken straight, and no fluids of any kind are allowed for at least 15 minutes after administration.

Supplied: In bottles of 3 fl.oz. and 16 fl. oz., at pharmacies everywhere

Kinney®

write for complete literature

KINNEY & COMPANY • COLUMBUS • INDIANA

Because so many parents
think of phenobarbital as a big, bad wolf . . .

. . . S.K.F. has dressed it up in sheep's clothing. 'Eskaphen B' Elixir is the ideal phenobarbital preparation for children. Its color, its taste, and its name completely disguise the phenobarbital content. When you write "Eskaphen B", anxious parents need not know you are prescribing a barbiturate.

ESKAPHEN B* Elixir & Tablets

(the disguised phenobarbital—with B_1)

Each 5 cc. teaspoonful of Elixir (and each tablet) contains:
phenobarbital, ¼ gr.; thiamine hydrochloride, 5 mg.

Smith, Kline & French Laboratories, Philadelphia

★T.M. Reg. U. S. Pat. Off.

When I become a doctor—I'll specify Clapp's Baby Foods for all my babies, too!

Copyright 1950, by Simon and Schuster, Inc.

The makers of Clapp's Baby Foods appreciate the fact that . . .

DOCTORS HAVE BEEN RECOMMENDING CLAPP'S BABY FOODS LONGER THAN ANY OTHER STRAINED BABY FOODS

CLAPP'S® BABY FOODS

Product of American Home Foods

KANANA BANANA FLAKES*

Pure Ripe Dehydrated Banana With Nothing Added

A FOOD OF CHOICE FOR INFANT FEEDING

1. Use as Baby's first solid food.
   Sweet tasting, willingly accepted, high food value.
2. Use in the dietetic management of celiac syndrome.
3. Use in the management of infantile diarrhea.*
4. Use as a milk modifier.
5. Use as a non-allergenic, nutritious food.**
6. Also—for malnutrition, ulcerative colitis and other gastrointestinal disturbances.

Fries, J. H., Chiara, N. J., and Waldron, R. J.: Dehydrated Banana in the Dietetic Management of Diarrheas of Infancy, J. Pediat., 37:367, 1950.

**Fries, J. H., and Glaser, I.: Studies on the Antigenicity of Banana, Raw and Dehydrated, J. Allergy, 21:169, 1950.*

ACCEPTED COUNCIL ON FOODS AND NUTRITION AM MEDICAL ASSOCIATION

Kanana BANANA Flake

★(NOT A POWDER)

FREE SAMPLES

Fill out and mail this coupon today to Kannengiesser & Company, Dept. JP, 76 Ninth Avenue, New York 11, N. Y. for content analysis and Free Samples of KANANA BANANA FLAKES.

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KANNENGIESSER & COMPANY, INC., 76 NINTH AVENUE, NEW YORK 11, N. Y.

Distributed in Eastern Canada by:
LAURENTIAN AGENCIES LIMITED
429 St. Jean Baptiste Street
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NATIONAL IMPORTERS L
418-20 East Pender St
Vancouver, B. C., Can

Upjohn

cortisone for inflammation, neomycin for infection:

Each gram contains:

Cortisone Acetate . . . . . 15 mg.

Neomycin Sulfate . . . . . 5 mg.

(equivalent to 3.5 mg. neomycin base)

Available in 1 drachm tubes with applicator tip

The Upjohn Company, Kalamazoo, Michigan

Neosone

Trademark

OPHTHALMIC OINTMENT

HURRY THAT SOOTHING AMMENS

My skin is irritated!

Prickly heat, diaper rash or other minor skin irritations respond rapidly to Ammens, for it discourages bacterial growth and protects macerated areas against moisture. Evenly dispersed in talc, the starch granules provide a maximum surface area for even absorption of moisture.

Its faintly medicinal odor makes Ammens Powder especially suitable for your professional recommendation.

Carefully formulated, combining starch, talc, zinc oxide, boric acid and hydroxyquinolin.

In Ammens Powder the starch granules seem to float in a sea of talc.

AMMENS®
MEDICATED POWDER

BRISTOL-MYERS COMPANY • 19 WEST 50 STREET, NEW YORK 20, N.Y.
Distributor for CHARLES AMMEN COMPANY • ALEXANDRIA, LOUISIANA

Reflects in your patients . . .

a fresh response and a vigorous improvement

armatinic activated

vitamin B_{12} plus activators

In Armatinic Activated, the hemopoietic factors activate and potentiate each other in their interrelated role in producing mature red blood cells.

THE ARMOUR LABORATORIES

A DIVISION OF ARMOUR AND COMPANY
CHICAGO 11, ILLINOIS

Each **ARMATINIC ACTIVATED** Capsulette contains:

Ferrous Sulfate, Exsiccated	200 mg.
*Crystamin	10 mcg.
Folic Acid	1 mg.
Ascorbic Acid (Vitamin C)	50 mg.
**Liver Fraction II (N. F.) with Desiccated Duodenum	350 mg.

*The Armour Laboratories Brand of Crystalline Vitamin B_{12}. **The liver is partially digested with duodenum during manufacture.

Supplied in bottles of 100 and 1000.

Also available: Armatinic Liquid, bottles of 8 oz. and 16 oz.

made over "live-foot" lasts...

Buster Browns are made to permit a child's normal foot to grow normally. They are made over "live-foot" lasts modeled from the feet of active, healthy children.

Preference for Buster Browns has been growing steadily for 49 years now because of the realistic way Buster Brown has followed nature's pattern. There is in every Buster Brown comfortable support and sensible growing room for children of every age. Buster Brown Division, Brown Shoe Company, St. Louis.

Buster Brown dealers always assure the correct size and proper fit for children. They take pride in this tested, proven method, which checks the feet 6 ways, giving all the vital points gentle support and room to flex and grow.

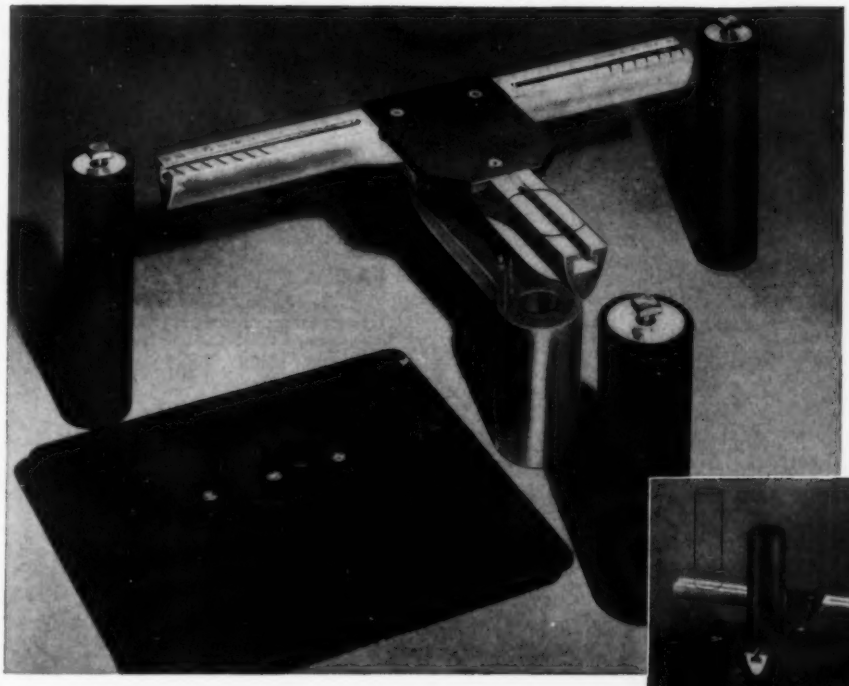
1. Both feet are measured, and the longer foot size and greater width is fitted.
2. Big toe joint is fitted to the widest inside line of the shoe.
3. Small toe is fitted to the widest outside line o. the shoe.
4. Adequate allowance from the end of the toe to the end of the shoe.
5. Heel fitted wide enough at bottom and snug enough at top.
6. Regular intervals suggested for rechecking shoe size.

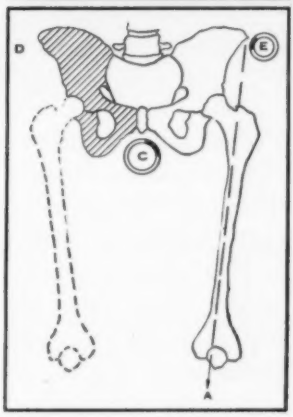
Buster Brown Shoes

AMERICA'S FAVORITE CHILDREN'S SHOES

New! PELVIC ANCHOR SACRAL REST*

...for unilateral traction





Castle introduces positive anchorage of the pelvis in orthopedic and traumatic surgical procedures. The Unit is adaptable to any major fracture table for use with child or adult patient to stabilize the pelvis in a centered position on the table.

AS ILLUSTRATED IN THE DIAGRAM, the Unilateral Traction Technique provides that when traction is applied to "A", counter-pressure is exerted at the ischium at "C" and on the ilium at "E". The combined counter-pressure at these two points of the innominate bone equals applied traction at "A". Tilting or pivoting of the pelvis is completely avoided—and of course, counter-traction on the opposite limb with all its attendant problems, is eliminated.

WRITE TODAY for complete information

70 YEARS OF PIONEERING DEVELOPMENT — Castle Sterilizers and Surgical Lights are recognized as the standard of engineering achievement and dependable performance throughout the world.

**WILMOT CASTLE CO. · 1198 University Ave. · Rochester 7, N. Y.*

**Hall, Custis, et al. Jour. International College of Surg.; pp 30-33, January 1952.*

new 3 year study[1] shows

"beneficial effect" of

DESITIN®

OINTMENT

the pioneer external **cod liver oil** therapy

in **extensive dermatitis, diaper rash, severe intertrigo, chafing, irritation** (due to diarrhea, urine, soaked diapers, etc.)

DESITIN OINTMENT achieved "significant amelioration" or practically normal skin in 96¾% of infants and children suffering intense edema, excoriation, blistering, maceration, fissuring, etc. of contact dermatitis. This and other recent studies recommend Desitin Ointment as "safe, harmless, soothing, relatively antibacterial"..... protective, drying and healing.[2-4]

Desitin Ointment is a non-irritant, non-sensitizing blend of high grade, crude Norwegian cod liver oil (with its high potency vitamins A and D, to benefit local metabolism,[1] and unsaturated fatty acids in proper ratio for maximum efficacy), zinc oxide, talcum, petrolatum, and lanolin. Does not liquefy at body temperature and is not decomposed or washed away by secretions, exudate, urine or excrements. Dressings easily applied and painlessly removed. Tubes of 1 oz., 2 oz., 4 oz.; 1 lb. jars.

samples and reprint[1] available from

DESITIN CHEMICAL COMPANY

70 Ship Street • Providence 2, R. I.

1. Grayzel, H. G., Helmer, C. B., and Grayzel, R. W.: New York St. J. M. 53:2233, 1953.
2. Helmer, C. B., Grayzel, H. G., and Kramer, B.: Archives of Pediatrics 68:382, 1951.
3. Behrman, H. T., Combes, F. C., Bobroff, A., and Leviticus, R.: Ind. Med. & Surgery. 18:512, 1949.
4. Turell, R.: New York St. J. M. 50:2282, 1950.

this child *could* gain 10 pounds

with **EDIOL** palatable oral fat emulsion

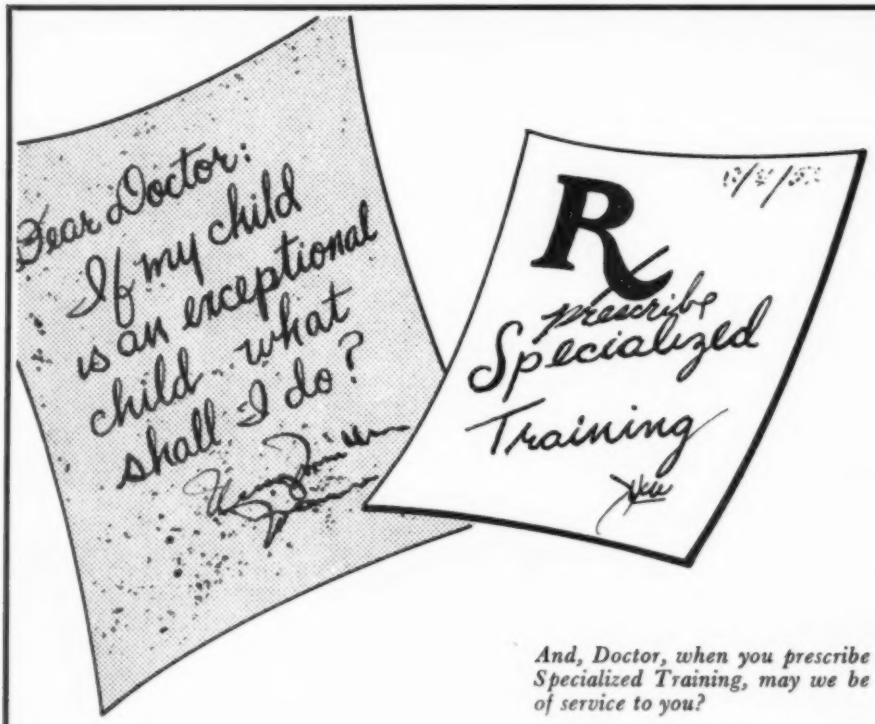
caloric boost

<u>without</u> gastric burden

An unusually palatable dietary additive, EDIOL can be taken alone and also combined with a variety of foods. Just two tablespoonfuls q.i.d. of this delicious oral fat suspension provide 600 extra calories. For still higher caloric intake, more may be prescribed as required.

EDIOL micronized emulsion of coconut oil (50%) and sucrose (12½%), supplied in bottles of 16 fluid ounces.

Schenley Laboratories, Inc.
New York 1, New York



And, Doctor, when you prescribe Specialized Training, may we be of service to you?

The Brown Schools is composed of seven separate residence centers, exclusive private schools offering specialized programs of education and re-education for children of all ages with educational and emotional problems.

Suburban and ranch locations in ideal year-round climate; our experienced resident staff of physicians, psychologists, and psychiatrists provides the ultimate in care and understanding of individual cases. Descriptive literature mailed on request.

Address inquiries to:
MRS. NOVA LEE DEARING, REGISTRAR
P.O. BOX 4008-D
AUSTIN, TEXAS

ACCEPTED FOR ADVERTISING IN PUBLICATIONS OF THE AMERICAN MEDICAL ASSOCIATION

The Brown Schools

BERT P. BROWN, PRESIDENT

Doctor, would it be helpful to you in your practice to know that there is a food available at reasonable prices in the stores the year round having these attributes:

1. One of the best "protective" foods with a well-rounded supply of vitamins and minerals.
2. Low sodium—very little fat—no cholesterol.
3. One of the first solid foods fed babies.
4. Useful in bland and low-residue diets.
5. Mildly laxative.
6. May be used in the management of both diarrhea and constipation.
7. Can be used in reducing diets.
8. Can be used in high-calorie diets.
9. Useful in the dietary management of celiac disease.
10. Useful in the dietary management of idiopathic non-tropical sprue.
11. Useful in the management of diabetic diets.
12. Valuable in many allergy diets.
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Every 4 to 6 hours

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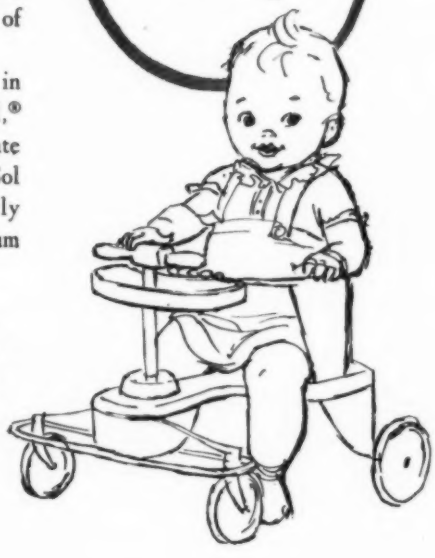

Iron deficiency anemia, "probably the commonest nutritional deficiency disease,"[1] occurs frequently in infants and children, particularly during periods of rapid growth.[2,3]

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(1) Youmans, J. B., in Handbook of Nutrition, Chicago, American Medical Association, 1951, p. 577; (2) Hansen, A. E., in Mitchell-Nelson Textbook of Pediatrics, ed. 5, Philadelphia, W. B. Saunders Co., 1950, p. 106; (3) Heck, F. J.: J.A.M.A. 148: 783, 1952.

0.6 cc. contains 75 mg. (about 1 grain) ferrous sulfate. Available in 15 and 50 cc. bottles with calibrated dropper.

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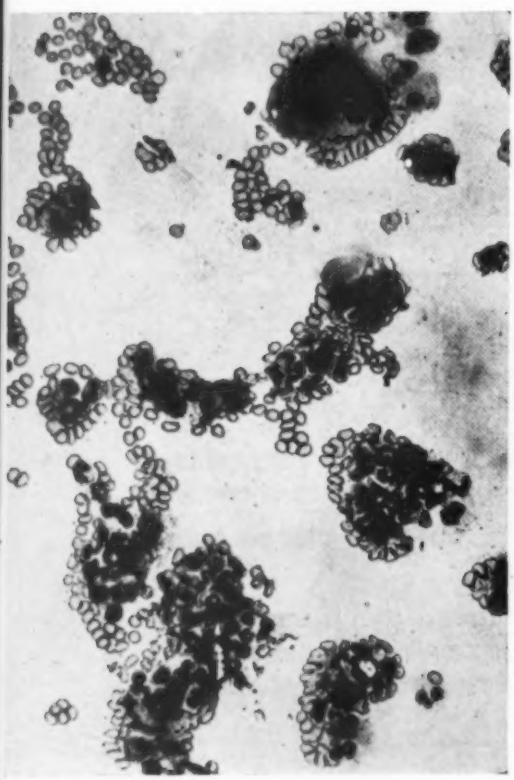
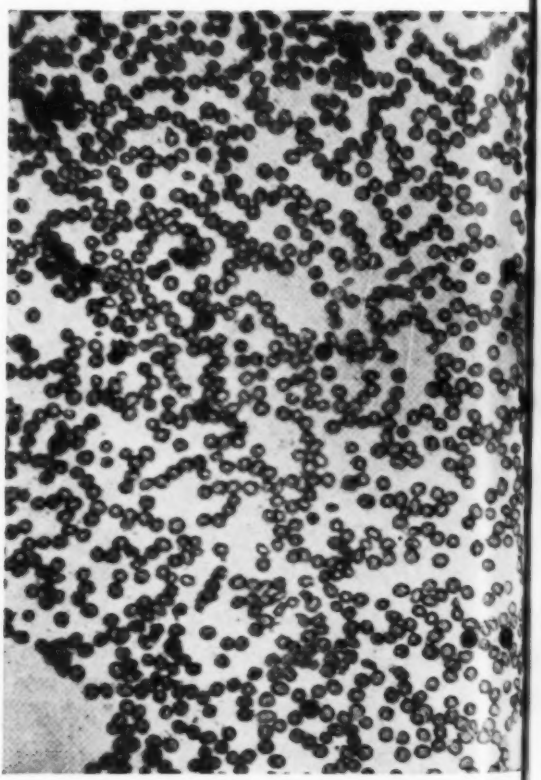
Because of extensive clinical studies with RONCOVITE—the original cobalt product—this understanding of direct stimulation of the depressed bone marrow has brought a completely new approach to the treatment of "secondary" anemia.



Bone marrow showing—acquired erythrocytic hypoplasia—no nucleated erythrocytes.



Same patient showing—active erythropoiesis following cobalt therapy.

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1. Case 2, Seaman, A. J., and Koler, R.; Acta Hematologica, 9:153, 1953.

2. Gardner, Frank H., J. Lab. Clin. Med.; 41:56, 1953.

3. Rohn, R. J. and Bond, Wm. H.; J. Lancet, 73:301, 1953.

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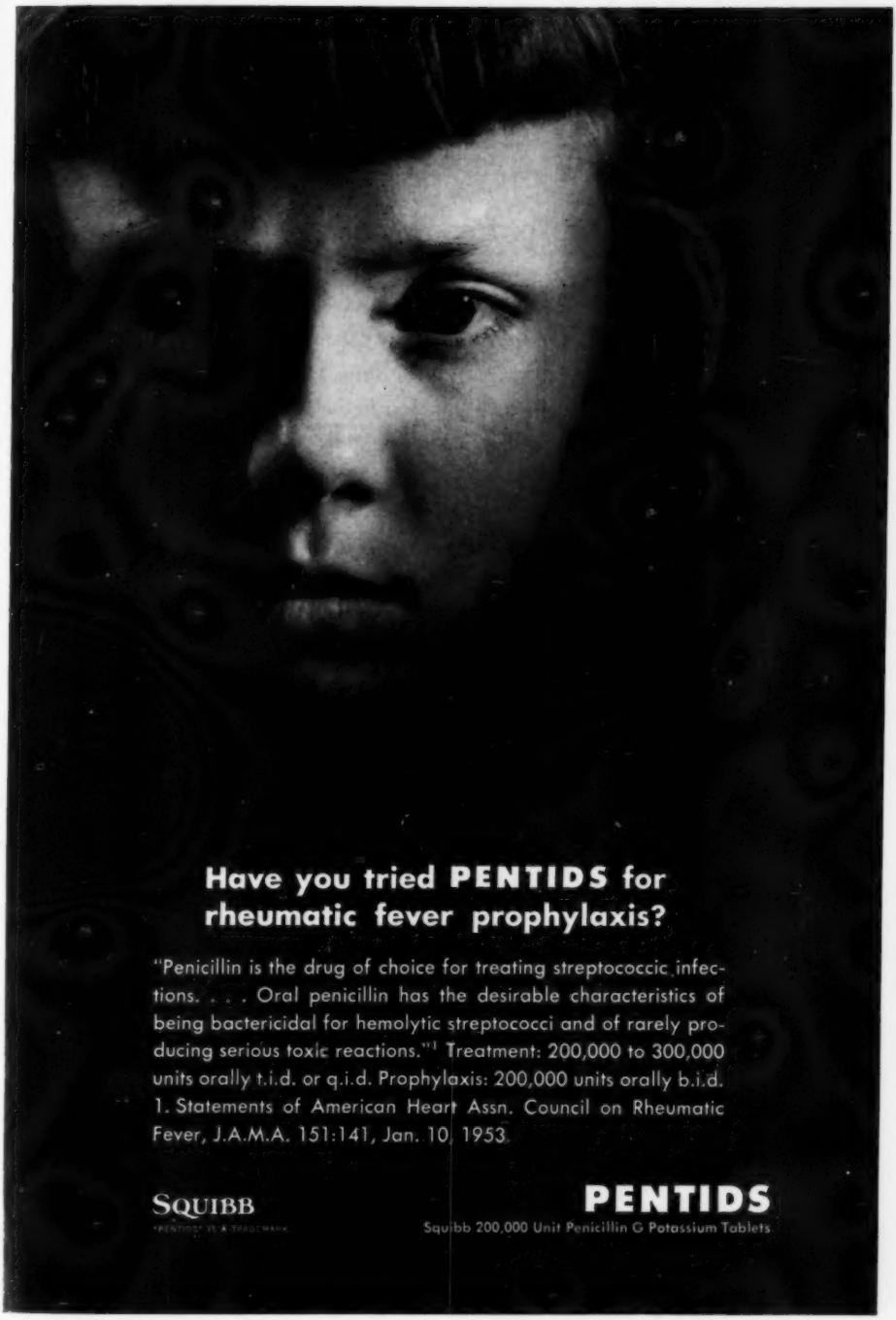
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1. Statements of American Heart Assn. Council on Rheumatic Fever, J.A.M.A. 151:141, Jan. 10, 1953.
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1. Boger, et al.: Oral Penicillin: Evaluation of All Forms, report distributed at New York Session of the A.M.A., 1953.

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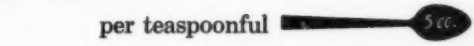
per teaspoonful 

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By Herbert Braunstein, M.D., Cincinnati, Ohio.

POLYPS OF THE RECTUM AND COLON IN CHILDREN.
By Reynold Erskine Church, M.D., and A. David Schwartz, M.D., New York, N. Y.

THE RELATIONSHIP OF OXYGEN CONSUMPTION TO CEREBRAL FUNCTIONAL ACTIVITY.
By Joseph M. Garfunkel, M.D., Henry W. Baird, III, M.D., and Joseph Ziegler, M.D., Philadelphia, Pa.

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By Byron B. Oberst, M.D., Omaha, Neb.

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By Louis Weinstein, M.D., Martin L. Vogel, M.D., and Norman Weinstein, M.D., Boston, Mass.

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STUDIES OF 17-HYDROXYCORTICOSTEROIDS. III. BLOOD LEVELS IN SALICYLATE INTOXICATION.
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FERROUS SULFATE POISONING. A PATIENT TREATED WITH BAL.
By Captain Joseph Shoss, Medical Corps, United States Air Force.

RUPTURED OMPHALOCELE COMPLICATED BY LACERATION OF THE STOMACH.
By Leo S. Bell, M.D., and Henry A. Brown, M.D., San Mateo, Calif.

CHYLOUS ASCITES.
By Clarence R. Laing, M.D., and Roland B. Scott, M.D., Washington, D. C.

THE ORIGIN OF OBSTETRIC NURSERIES.
By Stewart H. Clifford, M.D., Brookline, Mass., and Wilburt C. Davison, M.D., Durham, N. C.

HYPOTHERMIA IN CARDIAC SURGERY.
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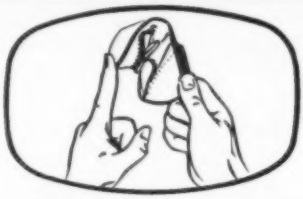

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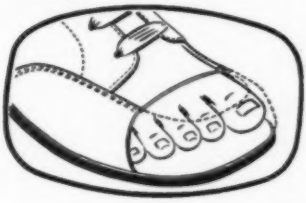

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†Editorial, J. Allergy 23: 279-280, 1952.

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CONTENTS FOR DECEMBER, 1953

Boric Acid Poisoning. Richard B. Goldbloom, M.D., and Alton Goldbloom, M.D., Montreal, Quebec ---- 631

Transcutaneous Absorption of Boric Acid. James Ducey, M.D., and D. Brooke Williams, M.D., New York, N. Y. ---- 644

Pertussis Immunization in Children With Convulsive Disorders. Karl-Axel Melin, M.D., Stockholm, Sweden ---- 652

Sickle-Cell Anemia Crisis. Emil Smith, M.D., Philip Rosenblatt, M.D., and Andrew V. Bedo, M.D., Brooklyn, N. Y. ---- 655

Some Problems in the Diagnosis and Treatment of Pulmonary Tuberculosis in Childhood. Han-Kuang Hsu, M.D., and John T. Szypulski, M.D. ---- 661

Congenital Paroxysmal Tachycardia. Ray C. Anderson, Ph.D., M.D., and Forrest H. Adams, M.D., Minneapolis, Minn. ---- 668

The Use of Hibicon in the Treatment of Epilepsy. Samuel Livingston, M.D., Baltimore, Md. ---- 673

Acute Gastroenteritis. Martin G. Wolfish, M.D., Toronto, Canada ---- 675

Single Ventricle With Diminutive Outlet Chamber. Rene P. Megevand, M.D., Raphael N. Paul, M.D., and Joseph Parker, M.D., Memphis, Tenn. ---- 687

Therapeutic Doses of Gamma Globulin in the Treatment of Measles Encephalitis and Encephalomyelitis. II. Cerebrospinal Fluid and Serum Studies in Measles Encephalitis and Encephalomyelitis With Special Reference to Phosphorus, Cholesterol, and Magnesium. Louis Odessky, M.D., Andrew V. Bedo, M.D., Philip Rosenblatt, M.D., Kenneth G. Jennings, M.D., Irving J. Sands, M.D., Harry Weisler, M.D., and Benjamin Newman, M.D., Brooklyn, N. Y. ---- 695

The Use of the Gardner-Murphy Pediatric Scalp-Vein Infusion Outfit. Pablo U. Abella, M.D., Boston, Mass. ---- 709

Generalized Cytomegalic Inclusion Disease. J. C. Bacala, M.D., and R. J. Burke, M.D., St. Louis, Mo. ---- 712

Treatment of Staphylococcic Pericarditis With Bacitracin. Max E. Freeman, M.D., and George F. Parker, M.D., Indianapolis, Ind. ---- 720

Primary Cardiac Tumors in Infancy. Luther A. Longino, M.D., and Irving A. Meeker, Jr., M.D., Boston, Mass. ---- 724

Medical Progress

A Current Summary of Strabismus in Children. Edme Regnier, M.D., Salem, Mass. ---- 732

Comments on Current Literature

Spread of Rickettsialpox. Russell J. Blattner, M.D., Houston, Texas ---- 740

News and Notes

News and Notes ---- 743

Book Reviews

Book Reviews ---- 745

Editor's Column

Boric Acid ---- 746

Should the Child With Convulsive Disorders Be Immunized Against Pertussis? 746

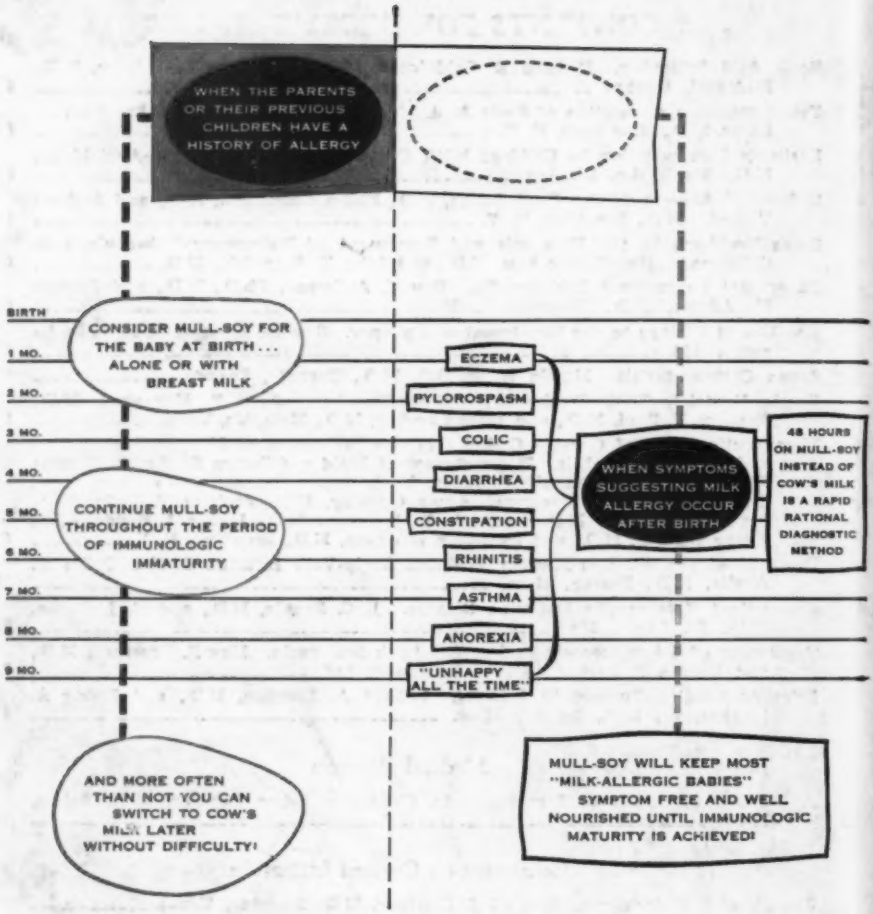
A Reported Increase in Accidental Poisoning in Children in Scotland ---- 750

Index

Index ---- 751

Current Pediatric References

Current Pediatric References ---- 55-C6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